March 1992
Volume 37, Number 3
ISSN 00989142-RECACP

RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
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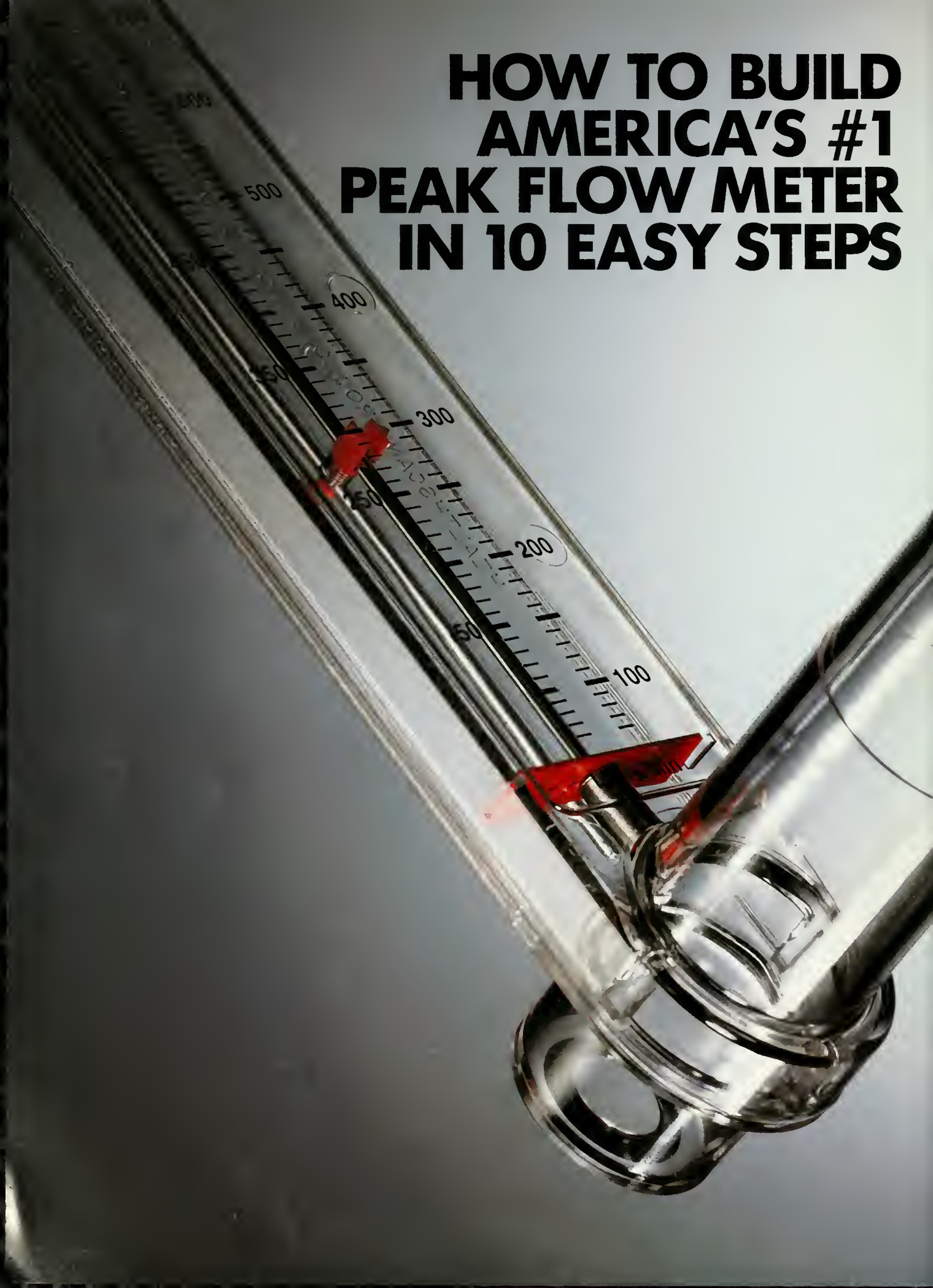
- Airway Hyperresponsiveness: What Can We Learn From RADS?
- Variations in Tidal Volume with Portable Transport Ventilators
- Transcutaneous and End-Tidal P_{CO_2} Monitoring
- Feasibility of FSV in Very Low Birthweight Infants
- Reactive Airway Dysfunction Syndrome: A Report of Three Cases
- An Approach to Metabolic Acidosis for the Respiratory Care Practitioner
- Respiratory Care Practitioner: Carpe Diem!



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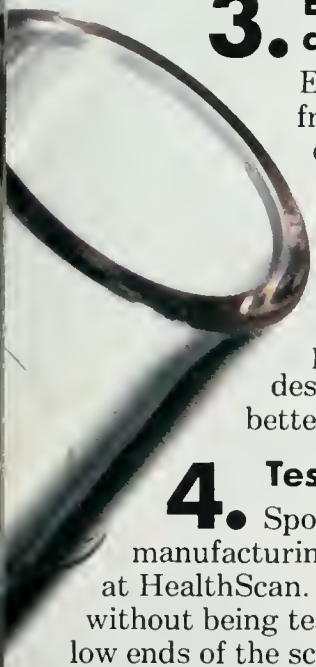

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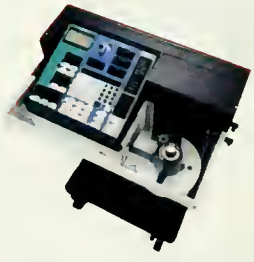

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Preparation of the Patient for Awake Flexible Fiberoptic Bronchoscopy (review). AP Reed. Chest 1992;101:244.

Newborn Screening for Cystic Fibrosis (Proceedings from the IVth International Conference on Newborn Screening for Cystic Fibrosis). JA Dodge, ed. Pediatr Pulmonol 1991;(7, Suppl):1-92.

The Impact of New Federal Regulations on the Blood Gas Laboratory (editorial). R Casaburi. Chest 1992;101:4-5.

A Controlled Trial of Synthetic Surfactant in Infants Weighing 1,250 Grams or More with Respiratory Distress Syndrome—W Long, A Corbet, R Cotton, S Courtney, G McGuiness, D Walter, et al. N Engl J Med 1991;325:1696.

BACKGROUND: Surfactant-replacement therapy is now recognized as a lifesaving and safe intervention in small premature infants, but there is little evidence concerning its risks and benefits in larger premature infants. METHODS: We conducted a placebo-controlled, blinded trial in 1,237 infants with respiratory distress who were enrolled at 23 hospitals in the United States and 13 hospitals in Canada. At entry all the infants weighed at least 1,250 g, were receiving mechanical ventilation, and had a ratio of arterial-to-alveolar-oxygen tension below 0.22. The initial dose of either the synthetic surfactant (Exosurf, 5 mL/kg of body weight) or air (the placebo) was administered less than 24 hours after birth, with a second dose given 12 hours later. A total of 614 infants were assigned to receive surfactant, and 623 to receive placebo. RESULTS: Fewer infants in the surfactant group than in the placebo group died before 28 days of age or survived at 28 days with bronchopulmonary dysplasia (7% vs 12%, p = 0.002). In the first 28 days of life, there were fewer deaths due to respiratory distress syndrome in the surfactant group (1% vs 3%, p = 0.043), lower overall neonatal mortality (4% vs 7%, p = 0.04), and a lower incidence of bronchopulmonary dysplasia (3% vs 6%, p = 0.008). There was also a significantly lower incidence of pulmonary air leaks, intraventricular hemorrhage, patent ductus arteriosus, seizures, hypotension, and pulmonary hypertension in the surfactant group. The infants treated with surfactant were weaned from oxygen and mechanical ventilation significantly sooner than those given placebo, and they less often required high-frequency ventilation or extracorporeal membrane oxygenation. The primary side effect observed more frequently among the infants who received surfactant treatment was pulmonary hemorrhage (6 infants vs 1 infant, p = 0.055). CONCLUSIONS: In infants weighing at least 1,250 g at birth who have respiratory distress syndrome, treatment with two doses of synthetic surfactant improves survival and reduces perinatal morbidity.

Hering-Breuer Reflex in Young Asthmatic Children—A Greenough, J Pool. Pediatr Pulmonol 1991;11:345.

The aim of this study was to assess if the Hering-Breuer reflex could be provoked in young children. Thirty asthmatic children with a mean age of 5.3 years and 18 healthy children with a mean age of 7.0 years were studied. The presence of the reflex was indicated by prolongation of the spontaneous expiratory time following application of continuous positive airways pressure (CPAP). The occurrence of the reflex was then related to age, compliance of the respiratory system (C_{rs}) measured by weighted spirometry, functional residual capacity (FRC) determined by helium gas dilution, and the change

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In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth.

Rescue

To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age.

CONTRAINDICATIONS

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WARNINGS

SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	SURVANTA (%)	Control (%)	*P* Value[a]
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	< 0.001
Pulmonary interstitial emphysema	20.2	38.4	< 0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a] *P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, *P* = 0.001, and 48.8% *v* 34.2%, *P* = 0.047, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

HOW SUPPLIED

SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0.9% sodium chloride solution. The color is off-white to light brown.

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June, 1991

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in lung volume resulting from the application of CPAP. The asthmatic children in whom the Hering-Breuer reflex was observed tended to be younger ($p < 0.01$) and the reflex was present in those children of both groups who had more compliant lungs ($p < 0.05$). The FRC was not different in asthmatic children with and without the Hering-Breuer reflex, but healthy children in whom the reflex was present tended to have smaller lung volumes. The change in lung volume resulting from application of CPAP was significantly greater in those children of both groups in whom the reflex was present ($p < 0.01$ asthmatics, $p < 0.02$ healthy children). We conclude that the Hering-Breuer reflex may be provoked in young children.

The Effect of Lung Mechanics on Gas Transport during High-Frequency Oscillation—M Schindler, M Seear. Pediatr Pulmonol 1991;11:335.

With the general aim of obtaining clinically relevant information on the use of high-frequency oscillation (HFO), we examined the effects of altering oscillatory frequency (f), tidal volume (V_T), and mean airway pressure (P_{aw}) on gas exchange in rabbits, both before and after altering the animals' pulmonary mechanics by saline induced lung injury. Twenty-seven combinations of f (5, 8, 12 Hz), V_T (0.5, 1, 2 mL/kg), and P_{aw} (5, 10, 13 cm H_2O) were used. Acute pulmonary injury was induced by instilling 10 mL/kg of warm saline into the lung. Gas exchange was assessed by steady-state levels of arterial oxygen tension (P_{aO_2}) and carbon dioxide tension (P_{aCO_2}). Arterial P_{aO_2} was independent of f or V_T before or after lung injury; it was independent of P_{aw} before injury but highly dependent on P_{aw} after lavage. The difference was presumably related to lung volume recruitment. Arterial P_{aCO_2} was dependent on f and V_T but independent of P_{aw} at any time. The relationship was modeled by the equation $P_{aCO_2} \propto f^a \cdot V_T^b$, where the exponents $a = -0.4$ and $b = -0.6$. Our technique of a standardized saline instillation gave a reproducible and stable model of lung injury. In damaged rabbit lungs the principles of HFO appear to be similar to conventional mechanical ventilation: oxygenation depends on P_{aw} and inspired oxygen concentration, while CO_2 removal is determined by f and V_T.

Effect of Prenatal Cocaine on Respiration, Heart Rate, and Sudden Infant Death Syndrome—JM Silvestri, JM Long, DE Weese-Mayer, GA Barkov. Pediatr Pulmonol 1991; 11:328.

We studied 114 neonates by pneumocardiogram recordings in order to examine the effects of cocaine with and without opiate exposure on neonatal respiration, heart rate, apparent life-threatening events (ALTE), and sudden infant death syndrome (SIDS). In full-term infants exposed to cocaine without opiates we found increased longest apnea duration and more episodes of bradycardia, but decreased periodic breathing and average heart rate than in control full-term infants. Term infants prenatally exposed to cocaine with opiates also had less periodic breathing. Preterm infants exposed to cocaine with and without opiates had decreased apnea density and periodic breathing compared with preterm controls. Discriminant analysis to determine whether perinatal asphyxia or exposure to other drugs could predict cardiorespiratory abnormalities showed no consistent relationship. In 72 of 114 infants available for follow-up, no ALTE occurred but two were lost to SIDS. Our data support the hypothesis that prenatal cocaine exposure may perturb, albeit subtly, the maturation of respiratory control, resulting in disruption of postnatal respiration.

Outcome of Home Care for Technology-Dependent Children: Success of an Independent Community-Based Case Management Model—Al Fields, DH Coble, MM Pollack, J Kaufman. Pediatr Pulmonol 1991;11:310.

Case management is important for successful home care of technology-dependent, respiratory-disabled children. Traditionally, the medical model of hospital-based home care and case management has been used for these children. The outcome may be different from when using independent, community-based home care and case management. We evaluated the results of 28 technology-dependent children (23 receiving mechanical ventilation and 5 receiving continuous positive airway pressure, CPAP) from 8 hospitals, who utilized an independent, community-based case management group to coordinate home care. After 26.3 ± 20.6 months of follow-up, 13 children (46%) remained technology-dependent, 10 (36%) were technology-independent, and 5 (18%), all with neurologic dysfunction, had died. Only one death was caused by a complication of technology. All children with congenital anomalies (n = 4), primary pulmonary disease (n = 8), and neuromuscular disease (n = 4) survived, and 9 (56%) were weaned from technological support. Children with chronic respiratory failure secondary to central neurologic dysfunction (n = 12) did poorly; 5 died, 6 remained technology-dependent, and only 1 became in-

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dependent of technology. Children with neuromuscular diseases tended to use less home care nursing at a lower home care cost. Parent satisfaction was high among those who responded (82%), indicating that the child, siblings, and family were better off with the child at home. These outcomes suggest that community-based home care and case management is a reasonable alternative to the hospital-based model.

Detection of Arterial Gas Embolism by Increased End-Tidal Nitrogen in Dogs—GB Russell, JM Graybeal, MT Snider. J Neurosurg Anesthesiol 1991;3:278.

Increased end-tidal (ET) nitrogen in a patient being ventilated with a nitrogen-free gas mixture through a leak-free circuit has been considered a specific sign of venous air embolism. We hypothesized that increased ETN_2 would occur after arterial air emboli, just as following venous air emboli, and that clinically relevant arterial air emboli could be detected with respiratory gas monitoring by mass spectrometry. After approval from the institutional Animal Utilization Committee, eight mongrel dogs were studied. All were anesthetized with pentobarbital and ventilated with room air by a volume ventilator. Each animal was monitored by a femoral artery and a pulmonary artery catheter for systemic and pulmonary blood pressures, respectively, an electrocardiograph, pulse oximetry, and inspired and expired respiratory gas measurements by mass spectrometry. Arterial blood gas analysis was undertaken after one series of air emboli. Air boluses (containing the nonradioactive nitrogen isotope $^{15}N_2$) of 50, 100, 200, and 500 μL/kg were injected slowly into the distal aorta through a second arterial catheter advanced 35 cm above the inguinal ligament. All emboli $\geq$ 100 μL/kg and 60% of the 50 μL/kg emboli were detected by increased $ET^{15}N_2$ within 30 s, reaching peak levels in < 2.75 min. The washout time for the $^{15}N_2$ was longer for larger emboli, ranging from 2.9 $\pm$ 2.8 min for 50 μL/kg emboli to 17.3 $\pm$ 3.2 min for the 500 μg/kg emboli. There were no significant changes in end-tidal carbon dioxide, pulmonary or systemic blood pressures, or arterial blood gases. Increased ETN_2 can no longer be considered pathognomonic for venous air embolism; arterial air embolism may have occurred.

The Effects of Postural Drainage and Positive Expiratory Pressure Physiotherapy on Tracheobronchial Clearance in Cystic Fibrosis—J Mortensen, M Falk, S Groth, C Jensen. Chest 1991;100:1350.

We studied the effects of two chest physiotherapy regimens on whole lung and regional tracheobronchial clearance (TBC) in 10 patients with cystic fibrosis. Their regimens were given on two separate days and consisted of 20 min of (1) postural drainage and the forced expiration technique (PD + FET), and (2) positive expiratory pressure (PEP mask) and FET (PEP + FET). A third day served as control. The study days were randomized. Each day, the clearance of lung radioactivity was measured for 3 h by gamma camera. The number of spontaneous coughs was recorded and the sputum expectorated was sampled. We found that both PD + FET and PEP + FET improved whole lung TBC at 30 minutes and 1 h four or fivefold ($p < 0.01$) compared with control, whereas at 2 h and 3 h only the improvement following PE + FET (approximately 1.4 times) was significant ($p < 0.05$). There was no significant difference in whole lung or regional TBC between the PD + FET and PEP + FET treatments. The correlations between TBC and the radioactivity content in sputum expectorated ($r^2 = 0.76$) and between TBC and numbers of coughs ($r^2 = 0.65$) were better than between TBC and the weight of sputum expectorated ($R = 0.39$). We conclude that PD and PEP when combined with FET have similar effects on short-term whole lung and regional TBC in patients with cystic fibrosis. Evaluation of TBC during chest physiotherapy when only based on the weight of sputum expectorated seems inadequate.

Extreme Variability in Aerosol Output of the DeVilbiss 646 Jet Nebulizer—MC Hollie, RA Malone, RM Skufca, HS Nelson. Chest 1991; 100:1339.

Ten new jet nebulizers (DeVilbiss 646) were studied to determine their consistency of output. Each nebulizer, containing 2 mL of saline solution, was run for 1 min to triplicate at four different straw (capillary tube) positions, keeping all other variables constant. Total output in milliliters per minute and volume of aerosol in the respirable range (1.0 to 5.0 μm) were measured. There was significant variability in output and volume of output in the respirable range (VORR) for every nebulizer at each of the tested straw positions ($p < 0.0001$). Irregularities in manufacture of the bowl and straw appear to be responsible for this significant variability. In summary, there is considerable intra-nebulizer and inter-nebulizer variability that could influence both patient care and medical use for diagnostic purposes.

Comparison of a Hand-Held Nebulizer with a Metered Dose Inhaler-Spacer Combination in Acute Obstructive Pulmonary Disease—GP Maguire, T Newman, LJ DeLorenzo, RB Brown, D Stone. Chest 1991;100:1300.

This study compared the effect of "standard" dose metaproterenol delivered by hand-held nebulizer (HHN) with two puffs of metaproterenol delivered by a metered dose inhaler (MDI) via a spacer (InspirEase) (MDI-spacer). Seventeen patients with an acute exacerbation of obstructive pulmonary disease were studied. Each patient received both MDI-spacer and HHN. Alternate patients were randomized to either MDI-spacer or HHN as initial treatment. Each subject was tested four different times: before and 30 min after the initial aerosol delivery technique. Testing consisted of spirometry, lung auscultation, and measurement of vital signs. The interval between treatments for all subjects was 2.96 ± 0.27 hours (mean $\pm$ SEM) and was not different for subjects who received therapy via MDI-spacer first or HHN first. The patient population studied demonstrated severe airways obstruction (baseline FEV_1 33.3% predicted $\pm 4.9\%$). There was a statistically significant improvement in FVC and FEV_1 after metaproterenol delivered by HHN, but not after MDI-spacer. Metaproterenol treatment with HHN resulted in a greater improvement in FEV_1 ($p < 0.05$) than MDI-spacer when the data were reported as absolute improvement (0.19 ± 0.05 L for HHN) vs (0.06 ± 0.03 L for MDI-spacer) or reported as percent change ($23.2 \pm 6.6\%$ for HHN) vs ($9.5 \pm 3.4\%$ for MDI-spacer). Asthmatic patients exhibited a significantly greater ($p < 0.05$) improvement in FEV_1 after HHN ($23.4 \pm 4.7\%$ change) than after MDI-spacer ($6.6 \pm 4.5\%$ change). Patients with chronic obstructive pulmonary disease (COPD) exhibited a greater improvement in FVC ($p < 0.05$) after HHN ($25.2 \pm 6.7\%$ change) than after MDI-spacer ($5.8 \pm 4.7\%$ change). We conclude that the "standard" dosage of metaproterenol delivered by HHN results in greater spirometric improvement in patients with acute obstructive pulmonary disease than the conventional dosage of metaproterenol delivered by MDI-spacer. It is likely that this reflects the fact that the recommended dose of metaproterenol delivered by MDI is too low and should be increased.

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Transcutaneous Oxygen Saturation and Carbon Dioxide Tension during Meals in Patients with Chronic Obstructive Pulmonary Disease—A Schols, R Mostert, N Cobben, P Soeters, E Wouters. Chest 1991;100:1287.

The effect on transcutaneous S_{aO_2} and trancutaneous carbon dioxide tension (P_{tcCO_2}) of eating was assessed in 44 patients with severe COPD ($FEV_1 < 50\%$). The S_{aO_2}, P_{tcCO_2}, and heart rate (HR) were measured every minute before, during, and until 30 minutes after a standardized meal (445 kcal) was consumed. All patients were measured twice on the same day, while eating a meal with high (80%) and low (28%) carbohydrate content, respectively. The mean meal desaturation (ΔS_{aO_2}), was less than 1% in normoxemic patients but was $-3.2 \pm 0.7\%$ ($p < 0.001$) in hypoxemic ($P_{aO_2} < 7.3$ kPa) patients. Significant differences between hypoxemic patients with a ΔS_{aO_2} greater than 4% and less than or equal to 4%, respectively, were found in FEV_1 ($16 \pm 3\%$ and $29 \pm 8\%$; $p < 0.001$), respiratory muscle strength (3.9 ± 1.2 kPa and 5.9 ± 1.2 kPa; $p < 0.01$), HR (112 ± 12 beats per minute and 90 ± 18 beats per minute; $p < 0.001$), body weight (54.9 ± 7.5 kg and 74.7 ± 10.4 kg; $p < 0.001$) and fat-free mass (42.0 ± 6.6 kg and 52.6 ± 5.8 kg; $p < 0.005$) but not in baseline S_{aO_2} and P_{tcCO_2}. The decrease in S_{aO_2} and the increase in HR were less during the carbohydrate-rich meal. No significant fluctuations in P_{tcCO_2} were found after either meal. Meal-related oxygen desaturation cannot explain weight loss in normoxemic patients with COPD but may contribute to a limited dietary intake in a subgroup of hypoxemic patients exhibiting marked oxygen desaturation during meals. A single carbohydrate-rich meal does not have an immediate impact on P_{tcCO_2} in stable COPD.

Pressure Support Ventilation with a Simplified Ventilatory Support System Administered with a Nasal Mask in Patients with Respiratory Failure—BE Pennock, PD Kaplan, BW Carlin, JS Sabangan, JA Magovern. Chest 1991;100:1371.

Thirty-one consecutive patients with respiratory failure in whom treatment with intubation and mechanical ventilation was being strongly considered received alternative ventilatory support by means of a BiPAP ventilatory support system and nasal mask. Laboratory measurements and physical findings were documented before and 1 hour after initiation of support. This support improved patient comfort, slowed respiratory rate, and improved oxygenation. Support lasted from 2 hours to 6 days. Seventy-six percent (22/29) of patients recovered from this episode of respiratory failure, avoiding alternative mechanical ventilatory support. There were no complications associated with aspiration, gastric distention, or acute separation from support.

Effects of Bicarbonate Therapy on Hemodynamics and Tissue Oxygenation in Patients with Lactic Acidosis: A Prospective, Controlled Clinical Study—D Mathieu, R Neviere, V Billard, M Fleyfel, F Wattel. Crit Care Med 1991;19:1352.

OBJECTIVE: To determine whether correction of acidemia using bicarbonate improves hemodynamic variables and tissue oxygenation in patients with lactic acidosis. DESIGN: Prospective, randomized, blinded, crossover study. Each patient sequentially received sodium bicarbonate and sodium chloride. The order of the infusions was randomized. PATIENTS: 10 patients with metabolic acidosis, increased arterial plasma lactate concentrations (> 2.45 mmol/L), and no severe renal failure (creatinine < 250 μ mol/L [< 2.3 mg/dL]). METHOD: Sodium bicarbonate (1 mmol/kg body weight) or equal volume of sodium chloride was injected I.V. at the beginning of 2 successive 1-h study periods. Period order was randomized. Arterial and venous blood gas measurements, plasma electrolytes (sodium, potassium, chloride), osmolality and lactate, 2,3-diphosphoglycerate (DPG), and oxygen hemoglobin affinity, hemodynamic variables, oxygen delivery, and oxygen consumption measurements were obtained before and repeatedly during the 1-h period after the injection of bicarbonate or sodium chloride. MEASUREMENTS & MAIN RESULTS: Sodium bicarbonate administration increased arterial and venous pH, serum bicarbonate, and the partial pressure of CO_2 in arterial and venous blood. Hemodynamic responses to sodium bicarbonate and sodium chloride were similar. Tissue oxygenation (as estimated by oxygen delivery, oxygen consumption, oxygen extraction ratio, and transcutaneous oxygen pressure) was not modified. No changes in serum sodium concentration, osmolality, arterial and venous lactate, red cell 2,3-DPG levels, or hemoglobin affinity for oxygen were observed. CONCLUSION: Administration of sodium bicarbonate did not improve hemodynamic variables in patients with lactic acidosis, but did not worsen tissue oxygenation.

Manual Resuscitators and Spontaneous Ventilation: An Evaluation—PJ Mills, J Baptiste, J Preston, GM Barnas. Crit Care Med 1991;19:1425.

BACKGROUND & METHODS: Although it is useful in certain clinical

situations for manual resuscitator units to be used with spontaneously ventilating patients, there are few data regarding their performance in these settings. We measured the percent-delivered oxygen from 13 adult manual resuscitator units during simulated spontaneous ventilation in the range of respiratory frequency, tidal volume, and oxygen supply in which manual resuscitator units might be used with patients. We also measured the resistive pressure developed during simulated ventilation and at constant inspiratory flow of 50 L/min. RESULTS: Oxygen supply, tidal volume, minute ventilation, and reservoir volume all influenced percent-delivered oxygen, but the most important determinant of percent-delivered oxygen was valve design. Valves incorporating a "disc" element to prevent air entrainment from the expiratory port gave the most efficient oxygen delivery, while "duckbill" valves did not reliably prevent air entrainment. Only two of the manual resuscitator units tested developed high resistive pressure. CONCLUSION: Reliable administration of high percent-delivered oxygen to spontaneously ventilating patients, while retaining the capability to manually ventilate them, is best achieved by a manual resuscitator unit with a valve of low resistance, incorporating a disc to prevent air entrainment. We recommend that manufacturers indicate on the product information sheet the degree (and confidence limits) to which their manual resuscitator unit presents resistance and delivers oxygen to a spontaneously ventilating subject.

Is Neonatal Inspired Gas Humidity Accurately Controlled by Humidifier Temperature?—M O'Hagan, E Reid, WO Tarnow-Mordi. Crit Care Med 1991;19:1370.

OBJECTIVE: To investigate (a) the relationship between humidifier temperature and inspired gas humidity and (b) the effect of insulating the inspiratory tube on "rainout" (condensate). DESIGN: Observational study. SETTING: Regional neonatal unit in a university hospital. PATIENTS: 48 infants receiving assisted ventilation, of whom 31 infants were nursed in incubators and 17 under radiant heaters. MEASUREMENTS & MAIN RESULTS: Despite always maintaining humidifier temperature > 34.7°C, inspired gas humidity decreased below the American National Standards Institution minimum of 30 mg H_2O/L on 35 of 479 occasions. At a humidifier temperature of 36°C, inspired gas humidity varied between 17 and 43 mg H_2O/L. In incubators set at a temperature of 34.1 ± 1.3 (SD) °C, inspired gas humidity was linearly related to humidifier temperature, but with wide scatter ($p < 0.001$, $r^2 = 0.28$). In cooler incubators set at 32.9 ± 1.8°C, inspired gas humidity varied inversely with humidifier temperature. This variation was attributed to condensate due to inspired gas cooling within the incubator. Insulation of the inspiratory tubing reduced condensate by only 15%. CONCLUSIONS: Inspired gas humidity cannot be predicted reliably from humidifier temperature. Accurate control will require a new generation of humidifiers that measure inspired gas humidity.

Survival from In-Hospital Cardiac Arrest with Interposed Abdominal Counterpulsation during Cardiopulmonary Resuscitation—JB Sack, MB Kesselbrenner, D Bregman. JAMA 1992;267:379.

OBJECTIVE: To determine whether interposed abdominal counterpulsation (IAC) during standard cardiopulmonary resuscitation (CPR) improves outcome in patients experiencing in-hospital cardiac arrest. DESIGN & SETTING: Randomized controlled trial in a university-affiliated hospital. PATIENTS: Patients experiencing in-hospital cardiac arrest during a 6-mo period. INTERVENTIONS: Patients were randomized to receive either IAC during CPR or standard CPR in the event of cardiac arrest. Abdominal compressions were performed during the relaxation phase of chest compression, corresponding to CPR diastole, at a rate of 80/min to 100/min. MAIN OUTCOME MEASURES: The three end points studied were (1) return of spontaneous circulation, (2) survival 24 hours after resuscitation, and (3) survival to hospital discharge. In addition, we examined neurological outcome in those patients surviving to hospital discharge. RESULTS: During the study period there were 135 resuscitation attempts in 103 patients. Return of spontaneous circulation was significantly greater in the group receiving IAC during CPR than in the group receiving standard CPR (51% vs 27%, $p = 0.007$). At hospital discharge, a significantly greater proportion of patients was alive in the IAC group than in the control group (25% vs 7%, $p = 0.02$). Eight (17%) of 48 patients who received IAC during CPR survived to hospital discharge neurologically intact, compared with only 3 (6%) of 55 patients from the standard CPR group (not significant). CONCLUSIONS: We conclude that the addition of IAC to standard CPR may improve meaningful survival following in-hospital cardiac arrest. The optimal use of this technique awaits further clinical trials.

Randomised Trial of Patient Triggered Ventilation versus High Frequency Positive Pressure Ventilation in Acute Respiratory Distress—MF Hird, A Greenough. J Perinat Med 1991;19:379.

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	Prophylactic treatment		Rescue treatment	
Age	*Single Dose*[2] 700-1100 grams (N=446)	*One vs Three Doses*[2] 700-1100 grams (N=716)	*Two Dose*[4] 700-1350 grams (N=419)	*Two Dose*[5] 1250 grams and above (N=1232)
≤10 Days	—	—	59%[§]	35%
≤28 Days	40%*	44%[‡]	52%[§]	39%
≤1 Year	44%[†]	41%[‡]	46%[§]	31%

*P=0.022. †P=0.002 ‡P<0.05. §P<0.001 ‖P 0.039 N=Number of infants enrolled in the clinical trials

A single prophylactic dose of EXOSURF Neonatal reduced 1-year mortality by 44%. Two additional prophylactic doses provided an additional 41% reduction in 1-year mortality.[3]

SIGNIFICANT REDUCTIONS IN MORTALITY FROM RDS IN MIDDLE-SIZE AND LARGE INFANTS[3,4]
(Percent reductions with EXOSURF)

	Prophylactic treatment	Rescue treatment	
	Single Dose[2] 700-1100 grams (N=446)	*Two Dose*[4] 700-1350 grams (N=419)	*Two Dose*[5] 1250 grams and above (N=1232)
Reduction in death from RDS	58%*	66%[†]	63%[‡]

*P=0.011 †P=0.007 ‡P=0.009 N=Number of infants enrolled in the clinical trials.

SIGNIFICANT REDUCTION IN CARDIOPULMONARY DESTRUCTION TO DAY 28 FOLLOWING TWO-DOSE RESCUE TREATMENT[4,5]

(Percent reductions with EXOSURF)

Birth weight	Reduction in death at 28 days or survival with broncho-pulmonary dysplasia (BPD)
700-1350[4] (N=419)	34%*
1250 and above[5] (N=1232)	43%*

*P=0.002. N=Number of infants enrolled in the clinical trials

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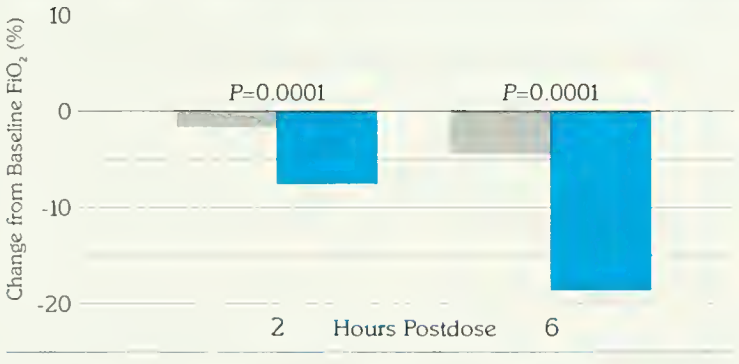
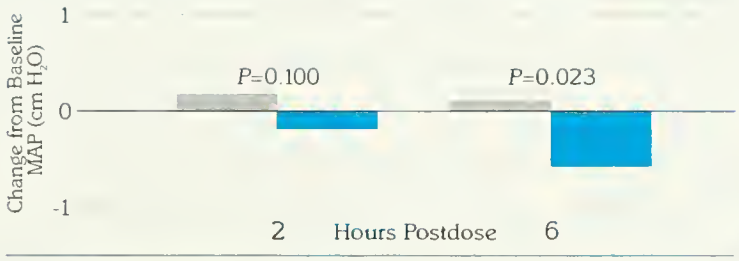
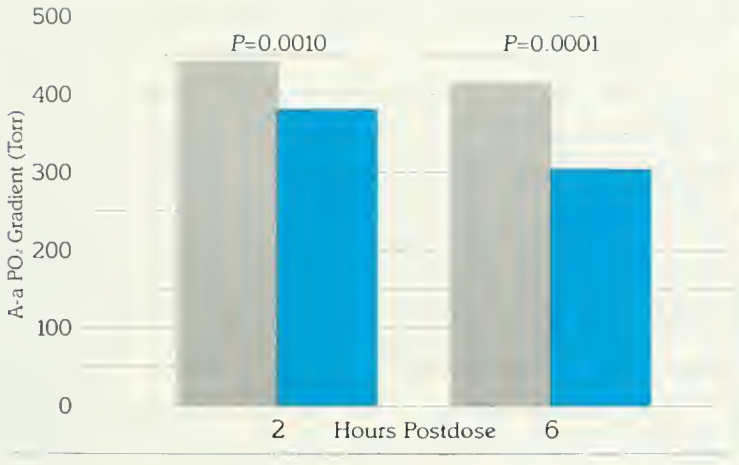
SIGNIFICANT IMPROVEMENTS IN SUPPLEMENTAL OXYGEN NEEDS AND VENTILATORY REQUIREMENTS SEEN IN MIDDLE-SIZE BABIES[4]







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One-year follow-up evaluated developmental outcomes[7]

Double-blind 1-year follow-up of more than 1450 infants enrolled in randomized trials showed that mental and motor scores appeared to be higher in tiny infants (<750 grams) as well as middle-size infants (750-1249 grams) who received Exosurf Neonatal.

Economic data analysis showed cost savings[8-10]

Three separate studies evaluated the economic impact of a single prophylactic dose of EXOSURF Neonatal, two-dose rescue treatment in 700- to 1350-gram infants, and two-dose rescue treatment during the neonatal period in infants weighing over 1350 grams. Results indicate that both prophylactic treatment and rescue treatment are cost-effective. Mean hospital charges were $6451 less for large infants receiving two-dose rescue treatment versus air in the first 28 days of life.[10]

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- **Easy to administer** Each EXOSURF Neonatal dose is administered in two 2.5-mL/kg half-doses without interrupting mechanical ventilation.
- **Easy on infant** To assist the distribution of EXOSURF Neonatal in the lungs, the infant is simply turned from midline position to the right after the first half-dose, and from midline position to the left after the second half-dose.

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INDICATIONS AND USAGE: Exosurf Neonatal is indicated for

1 **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS),

2 **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity and

3 **Rescue** treatment of infants who have developed RDS

CONTRAINDICATIONS. There are no known contraindications to treatment with Exosurf Neonatal

WARNINGS: Intratracheal Administration Only. Exosurf Neonatal should be administered only by instillation into the trachea. **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants. Instillation of Exosurf Neonatal should be performed **only** by trained medical personnel experienced in airway and clinical management of unstable premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal. **Acute Effects:** Exosurf Neonatal can rapidly affect oxygenation and lung compliance. **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak. **Hyperoxia.** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment. Failure to reduce FiO_2 in such instances can result in hyperoxia. **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow. **Pulmonary Hemorrhage.** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, pulmonary hemorrhage was reported for 1% (14/1420) of infants in the placebo group and 2% (27/1411) of infants in the Exosurf Neonatal group. Fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group. Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. In more than 7700 infants in the open, uncontrolled study, pulmonary hemorrhage was reported in 4%, but fatal pulmonary hemorrhage was reported rarely (0.4%). In the controlled clinical studies, Exosurf Neonatal treated infants who received steroids more than 24 hours prior to delivery or indomethacin postnatally had a lower rate of pulmonary hemorrhage than other Exosurf Neonatal treated infants. Attention should be paid to early and aggressive diagnosis and treatment (unless contraindicated) of patent ductus arteriosus during the first 2 days of life (while the ductus arteriosus is often clinically silent). Other potentially protective measures include attempting to decrease FiO_2 preferentially over ventilator pressures during the first 24 to 48 hours after dosing, and attempting to decrease PEEP minimally for at least 48 hours after dosing. **Mucous Plugs.** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately.

PRECAUTIONS:

General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry. However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered. The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection. **Prophylactic Treatment–Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS). Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups (see Table 1 in full product information). Data on multiple doses in infants in this weight class are not yet available. Accordingly, clinicians should carefully evaluate the potential risks and benefits of Exosurf Neonatal administration in these infants. **Rescue Treatment–Number of Doses:** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 µg/plate was not mutagenic in the Ames Salmonella assay. Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal. The effects of Exosurf Neonatal on fertility have not been studied.

ADVERSE REACTIONS

General: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died.

In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made. In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased. A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. Tables 3 and 4 summarize the results of the major safety evaluations from the controlled clinical studies.

Table 3: Safety Assessments[a]—Prophylactic Treatment

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N=108	EXOSURF N=107	Placebo (Air) N=193	EXOSURF N=192	Placebo (Air) N=222	EXOSURF N=224	EXOSURF 1 Dose N=356	EXOSURF 3 Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)								
Overall	51	57	31	27	36	36	38	35
Severe IVH	26	25	10	8	13	14	9	9
Pulmonary Air Leak (PAL)								
Overall	52	48	16	11	32	25	29	27
Pneumothorax	23	10*	5	6	19	11*	14	12
Pneumopericardium	1	4	2	0	<1	1	1	1
Pneumomediastinum	2	1	2	3	7	1**	3	2
Pulmonary Interstitial Emphysema	43	44	13	7*	26	20	23	22
Death from PAL	4	6	<1	<1	2	1	2	1
Patent Ductus Arteriosus	49	53	66	70	50	55	59	57
Necrotizing Enterocolitis	2	4	11	13	3	4	6	2*
Pulmonary Hemorrhage	2	10**	2	4	1	4	4	6
Congenital Pneumonia	4	4	2	4	2	2	1	1
Nosocomial Pneumonia	10	10	2	4	4	7	14	15
Non-Pulmonary Infections	33	35	34	39	28	29	35	34
Sepsis	30	34	30	34	23	24	30	27
Death From Sepsis	4	4	3	3	1	2	3	2
Meningitis	4	6	3	1	2	3	1	2
Other Infections	7	4	5	3	6	10	10	11
Major Anomalies	3	1	2	4	7	4	4	4

Table 3: Safety Assessments[a]—Prophylactic Treatment (Cont.)

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N=108	EXOSURF N=107	Placebo (Air) N=193	EXOSURF N=192	Placebo (Air) N=222	EXOSURF N=224	EXOSURF 1 Dose N=356	EXOSURF 3 Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
Hypotension	70	77	52	47	59	62	54	50
Hyperbilirubinemia	22	21	63	61	27	31	20	21
Exchange Transfusion	4	3	1	2	2	2	3	1
Thrombocytopenia[b]	21	25	not available		9	8	12	10
Persistent Fetal Circulation	0	1	1	1	0	2*	1	<1
Seizures	11	8	2	2	11	9	6	5
Apnea	34	33	76	73	55	65*	62	68
Drug Therapy								
Antibiotics	96	99	98	96	98	99	>99	99
Diuretics	55	60	39	37	59	63	64	65
Anticonvulsants	14	18	23	24	20	16	9	8
Inotropes	46	40	20	20	26	20	28	27
Sedatives	62	71	65	64	63	57	52	52
Pancuronium	19	11	22	14*	19	13*	15	11
Methylxanthines	38	43	77	77	61	72*	75	82*

[a] All parameters were examined with "as-treated" analyses. * p<0.05, ** p<0.01
[b] Thrombocytopenia requiring platelet transfusion

Table 4: Safety Assessments[a]—Rescue Treatment

Number of Doses / Birth Weight Range	Two Doses 700 to 1350 grams		Two Doses 1250 grams and above	
Treatment Group / Number of Infants	Placebo (Air) N=213	EXOSURF N=206	Placebo (Air) N=622	EXOSURF N=615
	% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)				
Overall	48	52	23	18*
Severe IVH	13	9	5	4
Pulmonary Air Leak (PAL)				
Overall	54	34***	30	18***
Pneumothorax	29	20*	20	10***
Pneumopericardium	4	1	1	2
Pneumomediastinum	8	4	5	2**
Pulmonary Interstitial Emphysema	48	25***	24	13***
Death from PAL	7	3	<1	1
Patent Ductus Arteriosus	66	57	54	45*
Necrotizing Enterocolitis	3	3	1	2
Pulmonary Hemorrhage	3	1	<1	1
Congenital Pneumonia	2	3	2	2
Nosocomial Pneumonia	5	7	2	2
Non-Pulmonary Infections	19	22	13	13
Sepsis	15	17	8	8
Death From Sepsis	<1	<1	1	<1
Meningitis	1	<1	1	<1*
Other Infections	5	8	5	6
Major Anomalies	3	3	4	4
Hypotension	62	57	50	39**
Hyperbilirubinemia	17	19	12	10
Exchange Transfusion	3	4	1	2
Thrombocytopenia[b]	10	11	4	<1**
Persistent Fetal Circulation	1	1	6	2**
Seizures	10	10	6	3*
Apnea	48	65**	37	44*
Drug Therapy				
Antibiotics	100	99	98	98
Diuretics	60	65	45	34***
Anticonvulsants	17	17	10	5**
Inotropes	36	31	27	16***
Sedatives	72	68	76	54***
Pancuronium	34	17**	33	15***
Methylxanthines	62	74**	49	53

[a] All parameters were examined with "as-treated" analyses. * p<0.05, ** p<0.01, *** p<0.001
[b] Thrombocytopenia requiring platelet transfusion

Pulmonary Hemorrhage. See WARNINGS. **Abnormal Laboratory Values.** Abnormal laboratory values are common in critically ill, mechanically ventilated, premature infants. A higher incidence of abnormal laboratory values in the Exosurf Neonatal group was not reported. **Events During Dosing.** Data on events during dosing are available from more than 8800 infants in the open, uncontrolled clinical study (Table 5).

Table 5: Events During Dosing in the Open, Uncontrolled Study[a]

Treatment Type / Number of Infants	Prophylactic Treatment N=1127	Rescue Treatment N=7711
	% of Infants	% of Infants
Reflux of Exosurf	20	31
Drop in O_2 saturation (≥ 20%)	6	22
Rise in O_2 saturation (≥ 10%)	5	6
Drop in transcutaneous pO_2 (≥ 20 mm Hg)	1	8
Rise in transcutaneous pO_2 (≥ 20 mm Hg)	2	5
Drop in transcutaneous pCO_2 (≥ 20 mm Hg)	<1	1
Rise in transcutaneous pCO_2 (≥ 20 mm Hg)	1	3
Bradycardia (<60 beats/min)	1	3
Tachycardia (>200 beats/min)	<1	<1
Gagging	1	5
Mucous Plugs	<1	<1

[a] Infants may have experienced more than one event.
Investigators were prohibited from adjusting FiO_2 and/or ventilator settings during dosing unless significant clinical deterioration occurred.

Reflux. Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes. ***Mucous Plugs.*** See WARNINGS.

DOSAGE AND ADMINISTRATION. See full product information.

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Synchronous respiration during mechanical ventilation of preterm neonates with acute respiratory distress is extremely beneficial as it improves oxygenation and is associated with a very low incidence of pneumothorax. We have assessed which form of ventilation—patient triggered ventilation (PTV) or high frequency positive pressure ventilation (HFPPV)—is most successful in provoking this beneficial respiratory interaction, synchrony. Preterm infants < 4 hours of age and gestational age ≥ 27 weeks were entered into a randomised controlled trial. Thirteen patients received PTV, median gestational age 30 weeks (range 27-36), and 36 HFPPV, median gestational age 29 weeks (range 27-40). HFPPV was delivered by Sechrist ventilators at rates between 61 and 120 breaths/min. Patient triggered ventilation was delivered by an SLE ventilator and an airway pressure trigger was used. Inflation times during PTV were between 0.2 and 0.45 seconds. HFPPV provoked synchrony that persisted until extubation in 25 patients, but PTV provoked persistent synchrony only in 4 patients ($p < 0.05$). No infant developed a pneumothorax. Eleven of 36 patients became asynchronous on HFPPV and 5 of 13 on PTV. In addition, 4 patients on PTV developed recurrent apnoea with deteriorating blood gases. Thus, 11 of 36 patients on HFPPV and 9 of 13 on PTV required transfer to conventional ventilation ($p < 0.05$). Transfer occurred at a median of 30 hours (range 6-84) on HFPPV and 1 hour (range 1-25) on PTV, $p < 0.01$. Infants who required transfer from the randomised mode of ventilation required a longer period of intubation (median 174 hours, range 30-2,928) compared to 38 hours (range 1.5-456) for successful cases, regardless of randomisation ($p < 0.01$). This study demonstrates PTV is significantly less successful in promoting synchrony than HFPPV. We therefore conclude HFPPV is a more useful form of respiratory support than PTV for preterm infants with acute respiratory distress.

Acute Effects of Maternal Cigarette Smoking on Fetal Heart Rate and Fetal Body Movements Felt by the Mother—LM Graca, CG Cardoso, N Clode, C Calhaz-Jorge. J Perinat Med 1991;19:385.

Acute effects of maternal cigarette smoking on fetal heart rate (FHR) and fetal body movements felt by the mother (FM) were studied in 51 pregnant volunteers. Thirty four were chronic smokers (6 or more cigarettes per day, with an average of 14 cigarettes/day) and 17 were sporadic smokers (1 to 5 cigarettes/day, with an average of 3 cigarettes/day). In both groups the number of FM, fetal reactivity, and short-term FHR variability decreased significantly in the 20 min following cigarette smoking; a sustained FHR rise of 10 or more beats/min was also found after the cigarette in more than 50% of the cases in the 2 groups. No statistically significant differences were found among the 2 groups when the post-cigarette data were compared. We conclude that maternal cigarette smoking produces important acute effects upon FM and FHR regardless of the average daily number of cigarettes smoked by the mother.

Induced Sputum and Cytological Diagnosis of Lung Cancer—RR Khajotia, A Mohn, L Pokieser, J Schalleschak, N Vetter. Lancet 1991; 338:976.

Diagnosis of lung cancer by examination of induced sputum specimens for malignant cells may be a valuable alternative to diagnosis by bronchoscopy. Patients suspected of having lung cancer were recruited and randomly distributed into two groups, one group (n = 34) having sputum induced by use of an ultrasonic nebuliser before bronchoscopy, and the other (n = 33) undergoing ordinary expectoration before bronchoscopy. 25 patients in the induced-sputum group were diagnosed as having primary lung cancer: induced sputum was positive for malignant cells in 21 of these patients (84%), whereas bronchoscopy was positive in 23 (92%) (not significantly different). In comparison, ordinary sputum was positive in 15 of 29 patients (52%) diagnosed as having primary lung cancer, whereas bronchoscopy was positive in 28 (97%) ($p < 0.001$). Induction of sputum by an ultrasonic nebuliser was an effective procedure for diagnosis of primary lung cancer.

The Pattern of Respiratory Muscle Recruitment during Pursed-Lip Breathing—EH Breslin. Chest 1992; 101:75.

Data from the present study indicate a change in the pattern of chest wall muscle recruitment and improved ventilation with pursed-lip breathing (PLB) in COPD. Pursed-lip breathing led to increased rib cage and accessory muscle recruitment during inspiration and expiration, increased abdominal muscle recruitment during expiration, decreased duty cycle of the inspiratory muscles and respiratory rate, and improved S_{aO_2}. In addition, PLB resulted in no change in pressure across the diaphragm and a less fatiguing breathing pattern of the diaphragm. Changes in chest wall muscle recruitment and respiratory temporal parameters concomitant with the increased S_{aO_2} indicate a mechanism of improving ventilation with PLB while protecting the diaphragm from fatigue in COPD. Alterations in the pattern of respiratory muscle recruitment with PLB may be

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associated also with the amelioration of dyspnea. Further investigation is necessary to explore the relationship between the pattern of respiratory muscle recruitment during PLB and dyspnea.

Incorrect Use of Metered Dose Inhalers by Medical Personnel—GG Guidry, WD Brown, SW Stogner, RB George. Chest 1992;101:31.

We administered a questionnaire and observed usage of a placebo metered dose inhaler (MDI) among 35 physicians, 14 nurses, and 12 respiratory therapists. Ninety-two percent of the respiratory therapists performed at least 4 of 7 steps correctly, compared with 65% of house staff physicians, 57% of nurses, and 50% of nonpulmonary faculty. Most participants followed package insert instructions, while only 18% followed recent recommendations for proper MDI use. We conclude that (1) medical personnel should have additional instruction in proper MDI usage and (2) respiratory therapists and nurses can play a prominent role in instructing patients in their proper use.

The Ventilator-Assisted Individual: Cost Analysis of Institutionalization vs Rehabilitation and In-Home Management—JR Bach, P Intintola, AS Alba, IE Holland. Chest 1992;101:26.

The purpose of this article is to present a cost analysis of in-home vs institutionalization for severely physically disabled ventilator-assisted individuals (VAIs). Following rehabilitation and adaptation to noninvasive methods of ventilatory support, 30 VAIs were maintained in the community for 12.9 ± 1.1 years with personal care attendants organized by a home care vendor reimbursed by New York City Medicaid. The program permitted self-directed severely disabled clients, including these 30 exclusively nontracheostomized VAIs, to live in the community and direct their attendant care and personal affairs. Prior to discharge home, the 30 patients resided in the respiratory unit of a long-term care facility for a mean of 8.9 ± 10.1 years. The unit is currently reimbursed at a mean rate of $718.80/patient/day. The current mean total cost of maintaining these VAIs in the community is $235.13 ± 56.73/patient/day. The conversion to and/or maintenance on 24-h nontracheostomy ventilatory support permitted discharge to the community by allowing the VAI to be attended by trained but uncredentialed home care attendants, thus avoiding prohibitively expensive in-home nursing for tracheostomy care. This created a savings to the public of 77% or $176,137/year/client. We conclude that conversion to and/or use of noninvasive methods of ventilatory aid can be a reasonable and cost-saving goal. More respiratory rehabilitation centers are needed to free up hospital beds and facilitate discharge of VAIs to the community. There is also evidence that trained attendants should be permitted to suction tracheostomized VAIs in the home.

Obstructive Sleep Apnoea in Patients with Dilated Cardiomyopathy: Effects of Continuous Positive Airway Pressure—S Malone, PP Liu, R Holloway, R Rutherford, A Xie, TD Bradley. Lancet 1991;338:1480.

The combined effects of negative intrathoracic pressure swings during obstructive sleep apnoeas (OSAs) and increased sympathetic nervous system tone associated with hypoxia and sleep arousal may lead to pulmonary oedema or left-ventricular hypertrophy. Therefore, we have done a study of patients with congestive heart failure secondary to idiopathic dilated cardiomyopathy to see whether OSA could contribute to impaired left-ventricular function and to assess nasal continuous positive airway pressure (NCPAP) for treatment. Eight men (aged 29-69 years) took part in the study; all were obese. Left-ventricular ejection fraction (LVEF) was measured while on stable medication and then 4 weeks after the start of nocturnal NCPAP. NCPAP was associated with abolition of OSA (mean [SE] number of apnoeas and hypopnoeas per hour of sleep 54.1 [7.2] and 1.0 [0.4] for pretreatment and NCPAP nights, respectively, $p < 0.0001$). Mean (SE) LVEF increased from 37 (4) % pretreatment to 49 (5) % after 4 weeks' NCPAP therapy ($p < 0.0001$). Withdrawal of NCPAP for 1 week in 4 patients was associated with a reduction in LVEF from 53 (6) % to 45 (5) ($p < 0.001$). OSA may contribute to impaired left-ventricular function in some patients with dilated cardiomyopathy of otherwise unknown origin, and reversal of OSA by NCPAP can lead to significant improvement in LVEF.

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Airway Hyperresponsiveness: What Can We Learn From RADS?

Airway hyperresponsiveness is an exaggerated bronchoconstrictor response to one or more of a variety of chemical and physical stimuli. The interaction between genetic and acquired factors in this basic feature of asthma is not entirely clear; however, there is strong evidence to implicate environmental influences in the pathogenesis of airway hyperresponsiveness.

Environmental influences on airway hyperresponsiveness can be insidious, often occurring over a long period of time, or they can be acute and dramatic. Subtle changes may occur following exposure to air pollutants such as ozone[1] or to acute infections with common viruses.[2,3] On the other hand, dramatic insults, similar to those described in the report appearing in this issue of RESPIRATORY CARE,[4] can also be associated with hyperresponsiveness. These often occur following accidental exposure to high concentrations of irritating agents and can lead to the reactive airways dysfunction syndrome (RADS).[5-11] Nasal manifestations from irritant exposure have also been described and recently termed reactive upper airway dysfunction syndrome (RUDS).[12]

There may be important similarities between the effects of acute and chronic insults to the airway. Bronchial hyperresponsiveness to nonspecific stimuli is felt to result in part from epithelial injury and chronic inflammatory changes of the airway.[13] Similar changes have also been observed in those few patients who have undergone biopsies following accidental irritant exposure. In their original description, Brooks and co-workers[5] suggested that the airway hyperresponsiveness of RADS is associated with an acquired and sustained altered threshold of airway irritant receptors and with increased epithelial permeability.[5] Our current understanding of the pathogenesis of asthma, which involves complex interactions of airway cells and mediators leading to inflammation,[13,14] overlaps with this theory of the pathogenesis of RADS. Cellular events accompanying the altered state of the airway may be common to both.

In summary, despite very different clinical scenarios, some basic similarities may be operating in the mechanisms of RADS and asthma that may help explain airway hyperresponsiveness in both illnesses.

Theodore J Witek Jr DrPH
E Neil Schachter MD

Departments of Pulmonary Medicine
& Community Medicine
The Mount Sinai School of Medicine
New York, New York

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Variations in Tidal Volume with Portable Transport Ventilators

Edward K McGough MD, Michael J Banner PhD, and Richard J Melker MD PhD

BACKGROUND: As intra- and interhospital transportation of ventilator-dependent patients has become more commonplace, the number of portable transport ventilators has increased. Transport ventilators should be capable of delivering consistent tidal volume (V_T) from breath to breath following changes in lung-thorax compliance and airways resistance. We sought to determine the effect of changes in compliance (C) and resistance (R) on the V_T delivered by eight commercially available, time-cycled transport ventilators. METHODS & MATERIALS: Each ventilator (PneuPAC Model 2, Autovent 3000, MAX, Bird Transport Mini-TXP, IC-2A, P7, E100i, and Logic 07a) was connected to a calibrated pneumotachograph and a test lung set for normal adult C (C = 100 mL/cm H_2O [1.02 L/kPa]) and R (R = 2 cm $H_2O \cdot s \cdot L^{-1}$ [0.2 kPa $\cdot s \cdot L^{-1}$]), with V_T at 1,000 mL. RESULTS: As C and R were manipulated, V_T varied widely. Tidal volume decreased least with the P7 and most with the Bird transport ventilator. CONCLUSION: Decreases in V_T with a transport ventilator predispose patients to hypoventilation, hypercapnia, and acidemia. Tidal volume often is not monitored continuously during transport, yet large decreases in V_T must not be allowed when pulmonary mechanics are unstable. Internal pressure-limiting valves, venturi flow-generating devices, and compression volume in the breathing circuit are at least three factors that affect V_T with transport ventilators. (Respir Care 1992;37:233-239.)

Background

With the increasing use of specialized diagnostic tests and therapeutic interventions for critically ill patients, the intrahospital transportation of ventilator-dependent patients has increased.[1] Moreover, the development of specialized centers, and the regionalization of care has led to frequent interhospital transportation of critically ill patients.[2,3] Frequently during transport, patients are ventilated by manual devices such as self-inflating bags or Elder demand valves; however, manual ventilation may not be practical for the long periods often required for transportation. The use of a mechanical transport ventilator also frees one member of the transport team for patient care and monitoring. Instability of critically ill patients during transport has been linked to the method of ventilation used during transport.[4-6] Because tidal (V_T) and minute volumes ($\dot{V}_E$), respiratory frequency (f), peak inflation pressure (PIP), and inspired oxygen concentrations (F_{IO_2}) may vary with manually operated devices, portable transport ventilators have been developed.[7-9] As the need for transport ventilators has increased, so have the number and complexity of these devices.[9] Some transport ventilators are designed for a specialized purpose, such as prehospital care, whereas others are designed to function as intensive care unit ventilators.

Dr McGough is associated with the Department of Anesthesiology, Dr Banner with the Departments of Anesthesiology and Physiology, and Dr Melker with the Departments of Anesthesiology, Emergency Medicine, and Pediatrics—University of Florida College of Medicine, Gainesville, Florida.

Presented in part at the Nineteenth Annual Educational and Scientific Symposium, Society of Critical Care Medicine, San Francisco, California, May 31, 1990.

No outside funding or material support was received for this study, and no financial arrangements exist between authors and manufacturers of the equipment studied.

The V_T, inspiratory flowrate, and airway pressure (P_{aw}) characteristics of transport ventilators under conditions of changing pulmonary mechanics have not been previously described. Transport patients who require mechanical ventilation often have abnormal pulmonary mechanics: Patients with pulmonary edema or adult respiratory distress syndrome have decreased lung compliance (C); patients with acute asthma or bronchospasm have increased airways resistance (R). Acute changes in C can occur secondary to conditions such as pneumothorax, hemothorax, or aspiration; mucus plugs, blood clots, and kinks in the endotracheal tube can all suddenly increase R. We hypothesized that, during transport, V_T may be adversely affected because of altered pulmonary mechanics, but time-cycled transport ventilators do not continuously monitor V_T. Therefore, we studied the effect of changes in C and R on V_T delivered by eight commercially available transport ventilators.

Methods and Materials

We evaluated the MAX, Autovent 3000, PneuPAC Model 2, Bird Transport Mini-TXP, IC-2A, P7 Model, E100i, and Logic 07a transport ventilators* (n = 2 for most models). All ventilators were operated according to manufacturers' instructions. No adjustable inspiratory pressure-limiting or 'pop-off' valves were readjusted during testing. The PneuPAC was operated with a 55-cm H_2O [5.39-kPa] pop-off valve; other interchangeable valves are available. Ventilators were powered by a 50-psig [344.75-kPa] oxygen supply. A calibrated pneumotachograph (Fleisch #2 with Gould-Statham differential pressure transducer) was positioned between the ventilator breathing circuit and a mechanical lung model. Airway pressure, flowrate, and V_T were displayed and recorded on a multichannel polygraph.

All ventilators were set to deliver a V_T of 1,000 mL [1 L]. To approximate normal adult values (control conditions), C was set at 100 mL/cm H_2O [1.02 L/kPa] and R at 2 cm $H_2O \cdot s \cdot L^{-1}$ [0.2 $kPa \cdot s \cdot L^{-1}$]. C and R were then varied to simulate increased R alone (C = 100 mL/cm H_2O [1.02 L/kPa], R = 20 cm $H_2O \cdot s \cdot L^{-1}$ [2 $kPa \cdot s \cdot L^{-1}$]), decreased C alone (C = 20 mL/cm H_2O [0.204 L/kPa], R = 2 cm $H_2O \cdot s \cdot L^{-1}$ [0.2 $kPa \cdot s \cdot L^{-1}$]), or both decreased C and increased R (C = 20 mL/cm H_2O [0.204 L/kPa], R = 20 cm $H_2O \cdot s \cdot L^{-1}$ [2 $kPa \cdot s \cdot L^{-1}$]). Ventilator settings were not adjusted following manipulations of C and R. The delivered V_T was recorded as the average of values from five consecutive breaths. All data were analyzed with a repeated measures ANOVA with a Tukey multiple comparisons test, and alpha was set at 0.05.[10]

Results

All ventilators functioned as described by the manufacturer (Table 1). Under control conditions, all ventilators delivered a V_T of 1,000 mL [1 L]

*Manufacturers and suppliers are identified in the Product Sources section at the end of the text.

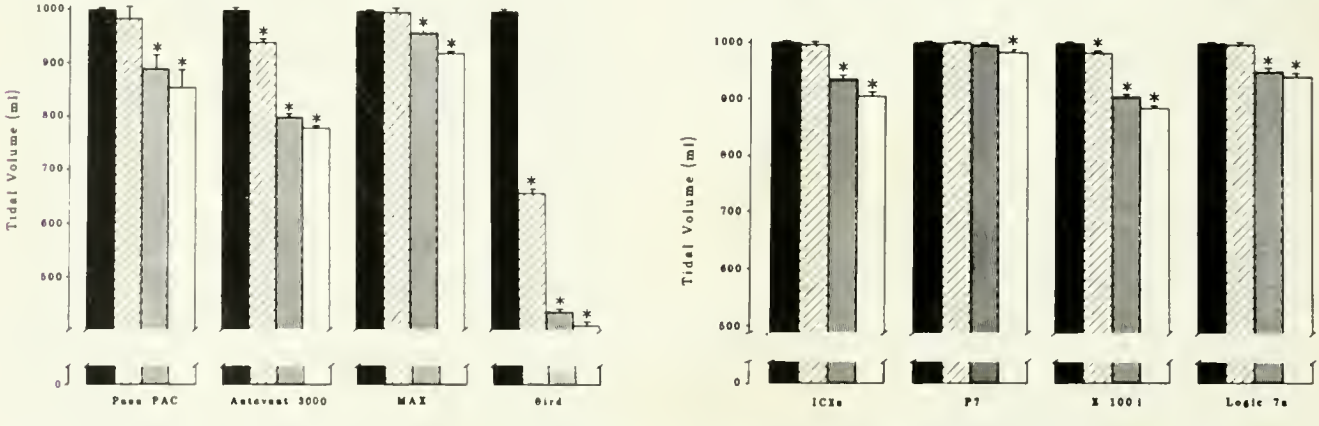

Fig. 1. Tidal volume delivered by eight transport ventilators at different levels of compliance (C) (mL/cm H_2O) and resistance (R) (cm $H_2O \cdot s \cdot L^{-1}$). Data are mean (SD); *$p < 0.05$ compared with control. R = 2, C = 100 for control ■; R = 20, C = 100 ▨; R = 2, C = 20 ▥; R = 20, C = 20 □.

Table 1. Characteristics of the Eight Transport Ventilators Studied

	Hamilton MAX	PneuPAC Model 2	Autovent 3000	Bird Mini-TXP	Bio-Med IC-2A	Newport E100i	Logic 07a	Bio-Med P7
Cost ($)	3,300	3,000	2,500	700	4,500	6,000	2,400	2,400
Ventilator weight (kg)	5	1.3	0.68	1.2	3.85	5.4	5	2.7
Gas consumption (L/min)	0.5	0.5	0.5	0	10	9	2	8
Manual breaths	Yes	No	No	Yes	Yes	Yes	No	No
Rate (breaths/min)	2-30	10-20	P: 9-27* A: 8-20	4-15	1-66	1-80	10-40	12-60
F_{IO_2}	1.0	0.45-1	1.0	0.45-0.8	1.0	0.21-1.0	0.5 or 1.0	1.0
Tidal volume (mL)	50-1,500	340-1,450	P: 200-600 A: 400-1,200	50-2,500	0-3,000	100-3,600	100-2,000	0-2,000
Peak mechanical inspiratory flowrate (L/min)	90	40	48	120	75	72	65	75
Cycling mechanism	Time	Time	Time	Time	Time	Time or pressure	Time	Time
Modes	IMV, CMV	CMV	IMV, CMV	CMV	CMV, IMV, CPAP	IMV, CMV, AMV, and CPAP	CMV	CMV, AMV
PIP Limit (cm H_2O)	60	55	45	Variable	≤ 100	≤ 80	70	100
PEEP (cm H_2O)†	No	No	No	No	0-25	0-25	No	No
Demand-flow Valve	Yes	No	Yes	No	No‡	No, continuous flow only	No	No
Airway pressure monitoring	Yes	No	No	No	Yes	Yes	Yes	Yes

* PEEP = positive end-expiratory pressure; PIP = peak inflation pressure; IMV = intermittent mandatory ventilation; CMV = controlled mechanical ventilation, CPAP = continuous positive airway pressure; AMV = assisted mechanical ventilation. P = Pediatric; A = Adult.

† PEEP can be applied with all ventilators by using an external PEEP valve.

‡ During spontaneous inhalation, the ventilator is patient-triggered "on," but the exhalation valve remains depressurized to allow venting of gas to the atmosphere. Depending on the inspiratory flowrate and time settings, gas flow for a specific duration is available for spontaneous breathing. The system does not function as a typical demand-flow system.

(Fig. 1). With a decrease in C and an increase in R, the P7 ventilator delivered the largest V_T (980 mL [0.88 L]) and the Bird Mini-TXP delivered the smallest (410 mL [0.4 L]) (Fig. 1); the remaining ventilators delivered a V_T greater than 850 mL [0.85 L] (85% of control) under the most severe condition (both decreased C and increased R), except the Autovent 3000, which delivered 780 mL [0.78 L]. With all ventilators under all conditions, f and inspiratory time (t_I) were unaffected. Under the

most severe condition, the pressure limits of the PneuPAC and Autovent 3000 were exceeded (55 and 45 cm H_2O [5.39 and 4.4 kPa], respectively). PIP for all ventilators ranged from 24 to 62 cm H_2O [2.35 to 6.08 kPa].

Discussion

The transport ventilators we tested functioned as either constant or nonconstant inspiratory flow generators. A constant flow generator delivers a constant inspiratory flow as PIP increases; with a nonconstant flow generator, a decelerating inspiratory flow results if PIP increases or is limited (Fig. 2). Because V_T equals inspiratory flowrate times inspiratory time, when inspiratory flow is constant and t_i is fixed, a preset V_T is delivered to the patient (assuming that the PIP limit is not exceeded and that compression volume in the breathing circuit is negligible). Conversely, if inspiratory flowrate decelerates (as with a nonconstant flow generator) and t_i is fixed, then V_T decreases.

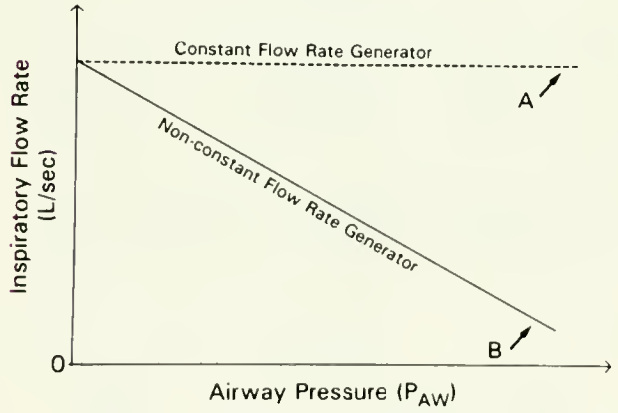

Fig. 2. Relationship between tidal volume (V_T) and airway pressure (P_{aw}) for transport ventilators that function as constant or nonconstant inspiratory flow generators. A constant flow generator delivers a constant inspiratory flow and, thus, constant tidal volume, as airway pressure increases (dashed line). A nonconstant flow generator delivers a decelerating inspiratory flow, and, thus, V_T decreases as P_{aw} increases (solid line).

A constant or square inspiratory flow waveform is generated under conditions of normal C and R and low PIP (Fig. 3). When C decreases and R increases, PIP increases; when P_{aw} equals the preset pressure limit, a decelerating inspiratory flow waveform results and V_T decreases. Under these conditions, in order to deliver a preselected V_T the PIP-limiting valve must be reset to a pressure greater than observed PIP. When the valve is reset, a higher PIP can be generated, which results in a constant inspiratory flow waveform and, thus, delivery of the preselected V_T.

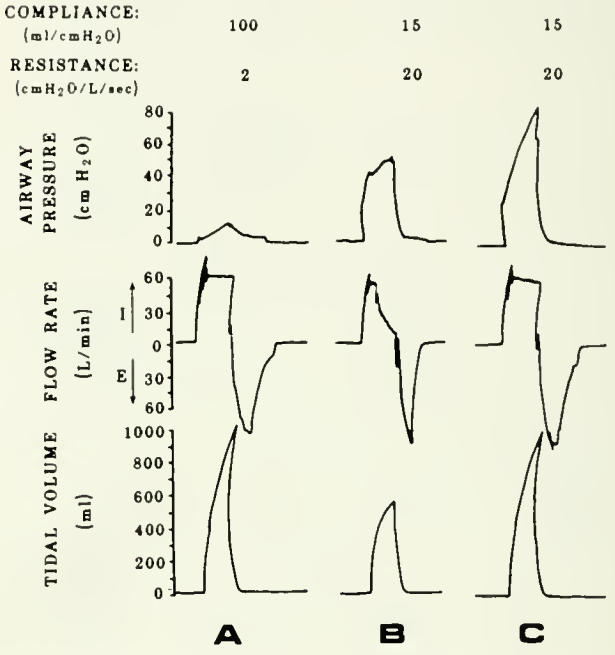

Fig. 3. Transport ventilators with a preset internal PIP-limiting valve deliver less tidal volume (V_T) when the limit of peak inflation pressure (PIP) is reached. Under conditions of normal compliance (C) and resistance (R), the PIP is less than the internal pressure limit and a constant inspiratory flow waveform and the preselected V_T (1,000 mL) are delivered (A). After a decrease in C and an increase in R, observed PIP increases to the internal preset pressure limit of the ventilator (50 cm H_2O [4.9 to 5.4 kPa] in this example), which results in a decelerating inspiratory flow waveform and a decrease in delivered V_T (B). When the internal pressure limit is reset above observed PIP, a nearly constant or square inspiratory flow waveform is again generated and the delivered V_T is restored to the preselected level (C).

Transport ventilators with venturi flow-generating devices are predisposed to deliver decreased V_T when pulmonary mechanics deteriorate. The amount of gas entrained into a venturi decreases, which decreases the rate of total outflow; thus, assuming a fixed t_i, V_T decreases as PIP increases. Under conditions of normal C and R, PIP is low, and a relatively constant inspiratory flow waveform and the preselected V_T are delivered

(Fig. 4). Following a decrease in C and an increase in R, however, PIP increases and the inspiratory flow waveform decelerates, which decreases V_T. Increasing the driving pressure into the venturi increases PIP, inspiratory flowrate, and V_T. Therefore, with transport ventilators that have a venturi flow-generating device, if pulmonary mechanics deteriorate, decreases in delivered V_T may not be detected unless V_T is monitored.

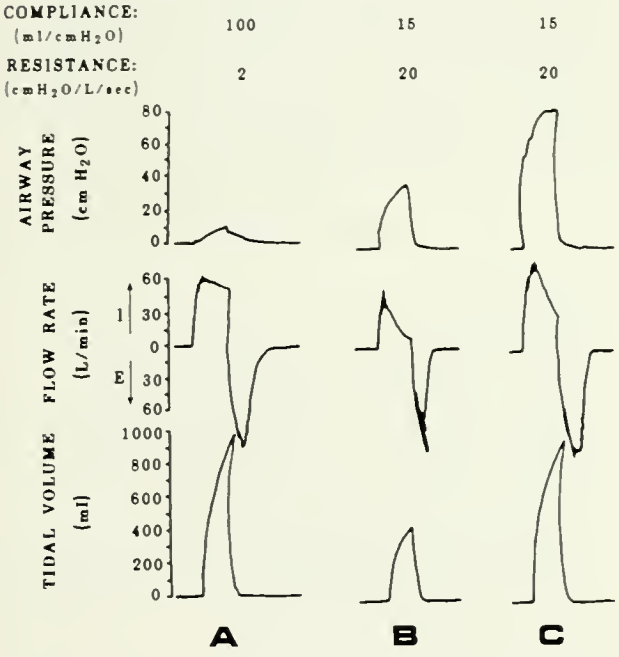

Fig. 4. Transport ventilators that use a venturi-flow-generating device (ie, Bird) are predisposed to variations in delivered tidal volume (V_T) as a result of changes in compliance (C) and resistance (R). Under conditions of normal C and R, peak inflation pressure (PIP) is low, a near constant or square inspiratory flow waveform is generated, and the preset V_T is delivered (A). After a decrease in C and an increase in R, PIP increases, which decreases flow entrained into the venturi, and, thus, in turn, a decelerating inspiratory flow waveform and a decrease in V_T result (B). Increasing the driving pressure into the venturi increases inspiratory flowrate and restores the delivered V_T to 1,000 mL [1 L] (C).

Transport ventilators should be able to generate enough inflating pressure to deliver the requisite V_T after an acute decrease in lung-thorax compliance or increases in R. Under either condition, a high PIP may be necessary to maintain alveolar ventilation and carbon dioxide elimination. However, PIP-limiting valves in transport ventilators preset by the manufacturer to a specific pressure limit may prevent the appropriate pressure from being generated and, thus, result in the delivery of a lower V_T to the patient. These pressure-limiting valves are intended to prevent barotrauma; in patients with very noncompliant lungs (such as those with adult respiratory distress syndrome), however, the risk of hypoventilation may exceed the risk of barotrauma. The control for the PIP-limiting valve should be both accessible and adjustable so that inflating pressure can be regulated according to the condition of the patient.

All transport ventilators tested in our study had a PIP-limiting valve except the Bird Transport Mini-TXP ventilator. The P7 and the Autovent 3000, for example, have maximum PIP limits of 100 and 45 cm H_2O [9.8 and 4.4 kPa], respectively. Because V_T equals the change in pressure times lung-thorax compliance ($\Delta P \times C$), as C decreases PIP must increase in order to deliver the preselected V_T. Similarly, as R increases, a greater inflating pressure is required to overcome the pressure drop across the resistance to maintain gas flow. Thus, it is not surprising that those transport ventilators that could generate a higher PIP delivered a higher V_T after changes in pulmonary mechanics.

The Bird Transport Mini-TXP ventilator has no PIP-limiting-valve mechanism per se. However, PIP is limited by regulating drive pressure (the inspiratory flowrate control knob on the ventilator) directed to the ventilator's venturi-flow-generating device. When P_{aw} generated during mechanical inhalation is in equilibrium with the preselected drive pressure to the venturi device, further increases in P_{aw} are precluded and the venturi stalls (ie, no gas is delivered to the patient). Plotting P_{aw} over time under this condition reveals a plateau-shaped airway pressure waveform. Increasing drive pressure increases the PIP limit. The maximal PIP attained is proportional to the drive-pressure setting. Conversely, lowering the drive-pressure setting results in a proportionately lower PIP limit.

Another factor that can affect V_T with a transport ventilator is the compression volume of the breathing circuit of the ventilator. Compression volume varies directly with the PIP generated during mechanical inhalation. For example, with a compression factor in the breathing circuit of 3 mL/cm H_2O [0.03 L/kPa] and a PIP of 10 cm H_2O

[0.98 kPa], approximately 30 mL [0.03 L] of the preset V_T are compressed within the breathing circuit, and the remainder is directed to the patient. If PIP increases to 50 cm H_2O [4.9 kPa], then the compression volume increases to approximately 150 mL [0.15 L] and the V_T delivered to the patient decreases proportionately. As compression factor and PIP increase, the breathing circuit compresses or 'traps' more volume and the patient receives less volume. Thus, the compression volume in transport ventilators should be as low as possible in order to minimize variation in delivered V_T.

Transport ventilators should be able to deliver a consistent $\dot{V}_E$. In an evaluation of accuracy of delivered $\dot{V}_E$ with transport ventilators, one ventilator delivered a $\dot{V}_E$ 95% greater than the set level.[11] Variation in $\dot{V}_E$ was noted in other ventilators as well. Because transport ventilators do not have flowsensors, substantial decreases in delivered V_T may not be detected under conditions of decreased C or increased R. As a result, patients are at risk from inappropriate $\dot{V}_E$ that may result in respiratory acid-base disturbances and cardiovascular instability. Continuous monitoring of end-tidal CO_2 (P_{etCO_2}) is another method used to assess whether the level of ventilation is appropriate. Currently P_{etCO_2} monitors are not portable enough to be used routinely during transport. Also, in patients with severe lung disease, the P_{etCO_2} may not adequately reflect arterial CO_2 (P_{aCO_2}).[12]

Conclusion

In order to deliver a preset V_T in spite of acute changes in pulmonary C and R, we recommend that transport ventilators meet the following criteria: (1) be able to generate a PIP adequate for the patient population being ventilated, possibly up to 120 cm H_2O [11.77 kPa] for critically ill patients (an upper limit has not been established); (2) to function as a constant inspiratory flow generator; (3) to have a PIP-limiting valve that can be adjusted; and (4) to have minimal gas compression volume in the breathing circuit. Of the ventilators tested, at the lowest compliance and highest resistance, the MAX, IC-2A, P7, and Logic 07a all delivered V_T within 10% of the control value. The PneuPAC and E100i were within 20% of control under the worst conditions tested. The Autovent 3000 and Bird Mini-TXP delivered a V_T less than 20% of control under the worst conditions of pulmonary mechanics. Because each ventilator has many different features, such as demand-flow valves, continuous positive airway pressure valves, and variable F_{IO_2}, no single ventilator can be recommended for all uses. We do not advise the use of ventilators that have a greater than 20% decrease in delivered V_T, unless exhaled V_T can be monitored. Further, we recommend that some method of assessing delivered V_T be readily available when any transport ventilator is used. The delivered V_T should be measured when ventilation is initiated, when ventilator settings are adjusted, and when there is a change in the patient's respiratory parameters such as PIP or breath sounds.

PRODUCT SOURCES

Ventilators:

MAX, Hamilton Medical Inc, Reno NV

Autovent 3000, Life Support Products, Irvine CA

PneuPAC Model 2, PneuPAC, Bedfordshire, UK

Bird Transport Mini-TXP, Bird Percussion Air, Sandpoint ID

IC-2A, Bio-Med Devices Inc, Madison CT

P7, Bio-Med Devices Inc, Madison CT

E100i, Newport Medical Instruments, Newport Beach CA

Logic 07a, Ohmeda, Columbia MD

Test Equipment:

Pneumotachograph, Fleisch #2 with Gould-Statham differential pressure transducer

Test Training Lung, TTL, Michigan Instruments, Grand Rapids MI

Multichannel polygraph, Grass Medical Instruments, Quincy MA

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Trancutaneous PCO_2 and End-Tidal PCO_2 in Ventilated Adults

Tim Blanchette MS RRT and John Dziodzio BA

Continuous noninvasive ventilation monitoring can provide valuable information for the management of the ventilated adult patient. We evaluated the accuracy of transcutaneous P_{CO_2} (P_{tcCO_2}) and end-tidal P_{CO_2} (P_{etCO_2}) measurements as compared to arterial CO_2 tension (P_{aCO_2}) measurements in adult patients on mechanical ventilators. METHODS: A convenience sample of 30 patients, ages 29 to 82 years, who were on mechanical ventilators in our Special Care Unit were included in the study. A P_{tcO_2}-P_{tcCO_2} sensor was placed on the upper chest of each patient. A mainstream P_{etCO_2} monitor-pulse oximeter was attached to the patient's artificial airway according to manufacturer's recommendations. Arterial blood samples were obtained and analyzed according to departmental protocol. RESULTS: 54 blood samples were obtained via indwelling catheters from the 30 patients (1-3 samples/patient) and P_{aCO_2} was compared to P_{etCO_2} and P_{tcCO_2}. The mean deviation (bias) for $P_{etCO_2} - P_{aCO_2}$ was –5.13 and the precision was 8.25 when all samples were compared. $P_{tcCO_2} - P_{aCO_2}$ produced an improved bias of –0.54 with a precision of 5.23 in the total population studied. In patients with documented respiratory disease, the 2-SD limits of agreement between P_{etCO_2} and P_{aCO_2} were –27.56 to 6.56 with a bias of –10.50 and a precision of 8.53. For $P_{tcCO_2} - P_{aCO_2}$ in this group of patients, the 2-SD limits of agreement were –14.11 to 13.70 with a bias of –0.21 and a precision of 6.95. In patients without respiratory disease, the limits of agreement were closer, bias did not differ between the two monitoring techniques, and the precision was not dramatically different. CONCLUSIONS: In patients with respiratory pathology, P_{tcCO_2} monitoring more accurately described P_{aCO_2} and provided a better indication of ventilation status than did P_{etCO_2}. In ventilated patients without respiratory disease, both monitoring techniques were similarly accurate. These findings point to the importance of assessing patient pathology and instrumentation when instituting and utilizing noninvasive ventilation monitoring techniques. (Respir Care 1992;37:240-248.)

Introduction

Noninvasive monitoring of carbon dioxide (CO_2) can provide an indication of the adequacy of ventilation in the ventilated intensive care patient. Transcutaneous P_{CO_2} (P_{tcCO_2}) monitoring utilizes a warmed Severinghaus electrode attached to the skin to provide a measurement of CO_2 tension at the skin surface. Common end-tidal CO_2 (P_{etCO_2}) monitors use a sampling adapter at the patient airway to collect exhaled gas and employ infrared wavelengths of light to determine the CO_2 percentage (or tension). There are advantages and disadvantages to the use of either end-tidal CO_2 (more correctly termed peak-expired CO_2) or transcutaneous CO_2 (P_{tcCO_2}) monitoring in the critically ill adult. Increased dead space and ventilation/perfusion ($\dot{V}/\dot{Q}$) inequities have been shown to affect the correlation between P_{etCO_2} and P_{aCO_2} in various patient populations.[1-8] Transcutaneous P_{aCO_2} monitoring is well established in neonates,[9-16] but has been infrequently used in the adult ICU

Mr Blanchette is a Respiratory Therapist and Mr Dziodzio is a Systems Analyst, Division of Pulmonary Medicine, Maine Medical Center, Portland, Maine.

This study was completed at the Maine Medical Center, Portland, Maine, and a version of this paper was presented at the RESPIRATORY CARE OPEN FORUM, during the 1990 AARC Annual Meeting in New Orleans, Louisiana.

patient.[17,18] To our knowledge, there are no studies that have compared these two monitoring techniques in ventilated adult intensive care patients suffering from a variety of diseases. We compared P_{etCO_2} and P_{tcCO_2} values to P_{aCO_2} in such a group.

Materials and Methods

Population and Sample

The convenience sample consisted of 30 patients ages 29 to 82 years (mean age = 58.7 y) with a mean weight of 81.5 kg (range 50-139 kg). All patients required ventilatory support, had indwelling arterial catheters, and were intubated and attached to mechanical ventilators. Patients were divided into two predetermined groups depending on the absence or presence of pulmonary pathology. Ten patients had recently undergone open heart surgery. None of the 10 suffered from respiratory disease based on history or diagnostic data. Five patients suffered from intracranial neurologic damage without lung involvement. Patients were often on vasoactive medications. Fifteen of the 30 patients (including 4 patients with adult respiratory distress syndrome, ARDS) exhibited abnormal pulmonary physiology that could have resulted in increased dead space and ventilation-perfusion mismatching. Patient data are presented in Table 1.

Study Protocol

A SensorMedics Transcend (SensorMedics Corporation, Anaheim CA) combination transcutaneous P_{O_2}-P_{CO_2} monitor was used to obtain P_{tcCO_2} values. The sensor membrane was new, the portable Transcend shuttle battery was fully charged, and the monitor was calibrated at the beginning of each day of monitoring. The sensor was placed on the upper chest of each patient with the sensor temperature set at 44°C. A 20-minute warmup time was observed before any data were collected. A Novametrix 7000 (Novametrix Medical Systems, Wallingford CT), which is a combination infrared mainstream P_{tcCO_2} monitor and pulse oximeter, was calibrated and attached to the patient's artificial airway according to manufacturer's recommendations. After a minimum of 20 minutes, and as departmental protocols dictated, blood samples from the indwelling arterial catheter were drawn for blood-gas analysis (ABG). P_{etCO_2} readings were recorded at the time the blood samples were drawn. The maximum P_{etCO_2} value was recorded if values varied with respiratory pattern (as during IMV). P_{tcCO_2} readings were recorded immediately after the blood samples were drawn. All blood samples were analyzed within 10 minutes (AVL 945 blood gas analyzers, AVL Scientific, Gras, Austria). ABG quality control procedures include the analysis of three levels of ABG controls and four levels of precision buffers every 12 hours, with blood comparisons and random controls every 4 hours. The blood-gas laboratory is enrolled in the College of American Pathologists and Accutest Proficiency Testing Programs, which provide unknowns quarterly for analysis. A total of 54 data sets were obtained from the 30 patients. From 1-3 data sets (consisting of P_{aCO_2}, P_{etCO_2}, and P_{tcCO_2}) were collected from each patient. P_{etCO_2} and P_{tcCO_2} values were each paired with P_{aCO_2} in the 54 samples, and the differences in the paired data were calculated by subtracting the P_{aCO_2} from the P_{tcCO_2} or P_{etCO_2}. All ABG values were corrected to the patient's body temperature before they were recorded. Pulse oximeter saturations and P_{tcO_2} values were ignored for the purposes of this study.

Statistical Analysis

We used the method of Bland and Altman[19] for analyzing bias and precision and determining the limits of agreement between the methods of clinical measurement.

The differences in the paired data were calculated by subtracting the P_{aCO_2} from the corresponding transcutaneous or expired CO_2 value. The bias between the techniques was assessed by calculating the mean of these differences. Precision is calculated as the standard deviation of the differences. Assuming the differences are normally distributed, 95% of the differences should appear within the range of bias $\pm$ 1.96 $\times$ precision. For simplicity, we use 2 $\times$ the precision in the calculation, which defines the limits of agreement between techniques.

Results

The limits of agreement were calculated for the total group and for those with and without respiratory disease. The bias of each monitoring technique was assessed by calculating the mean of the

Table 1. Variables Related to Diagnosis, Age, Ventilation, and Blood Pressure in 30 Study Patients

Patient	Diagnosis	Age	Ventilator				Mean Arterial Pressure (cm H_2O)
			Mode	f	PEEP	$\dot{V}_E$ (L)	
Patients with Respiratory Disease							
1	ARDS, multisystem failure	80	A/C	37	20	23	63
2	ARDS, pneumonia, pneumothorax	37	A/C	18	7.5	16	115
3	ARDS, right upper lobectomy	59	A/C	30	15	26	75
4	Resolving ARDS, urinary infection, pneumothorax	52	3 PS		5 (CPAP)	15	69
5	COPD, exacerbation	74	A/C	8	—	—	78
6	COPD, pneumonia	82	A/C	12	—	—	59
7	COPD, duodenal ulcer repair	61	IMV	4	—	7.0	80
8	Severe emphysema, pneumonia, sepsis	61	IMV	4	—	6.6	85
9	Respiratory failure, obesity, alveolar hypoventilation	73	IMV 5 PS	3	5 —	9.2 —	104 —
10	Respiratory failure, liver resection, renal failure	78	IMV	12	5	10.8	56
11	Respiratory failure, duodenal ulcer repair	53	A/C	—	—	7.1	78
12	Congestive heart failure, pneumonia	65	A/C	8	3	6.4	—
13	Pulmonary edema, post cardiac and respiratory arrest	68	A/C	8	—	9.0	75
14	Pulmonary emboli, respiratory failure	47	A/C	14	5	16.8	—
15	Restrictive lung disease, tracheotomy, sigmoid resection	72	CPAP	—	—	—	85
Patients without Respiratory Disease							
16	Subarachnoid hemorrhage	51	IMV	7	—	12.6	78
17	Closed head injury, multiple trauma	43	IMV	14	5	11.8	99
18	Closed head injury	53	A/C	—	—	9.6	82
19	Closed head injury	29	A/C	—	—	11.0	91
20	Closed head injury	44	IMV	5	5	—	—
21	CABG	61	IMV	6	—	—	—
22	CABG	62	A/C	8	5	8.8	68
23	CABG	77	A/C	9	—	6.3	74
24	CABG	69	A/C	8	—	6.4	70
25	CABG	41	A/C	8	5	8.0	77
26	CABG	69	A/C	10	—	7.7	83
27	CABG	65	A/C	8	5	8.0	93
28	CABG	75	A/C	8	—	8.0	81
29	Aortic valve replacement	65	A/C	10	—	7.5	86
30	CABG, aortic valve replacement	72	A/C	9	5	9.0	79

* $\dot{V}_E$ = minute ventilation (L); A/C = assist/control or controlled mechanical ventilation (CMV) (Puritan-Bennett 7200); IMV = intermittent or synchronized intermittent mandatory ventilation; PS = pressure support.

differences between the paired data. With all samples included, the bias for P_{tcCO_2} vs P_{aCO_2} was minimal and P_{etCO_2} vs P_{aCO_2} was negative and substantial (Table 2). Also, as seen in Table 2, transcutaneous monitor bias remained minimal both in patients who had respiratory disease and in those

Table 2. Summary of Bias, Precision, and Limits of Agreement*

	P_{tcCO_2} vs P_{aCO_2}			P_{etCO_2} vs P_{aCO_2}		
	Bias	Precision	LOA	Bias	Precision	LOA
All patients (n = 30)	–0.54	5.23	–0.54 ± 10.46	5.13	8.25	–5.13 ± 16.50
Patients free of respiratory disease (n = 15)	–0.80	3.39	–0.80 ± 6.78	–0.83	4.87	–0.83 ± 9.74
Patients with respiratory disease (n = 15)	–0.21	6.95	0.21 ± 13.90	–10.50	8.53	–10.5 ± 17.06
Patients with ARDS (n = 4)	0.44	9.17	0.44 ± 18.33	–18.44	5.29	–18.44 ± 10.5

* Bias = mean of the differences ($P_{etCO_2} - P_{aCO_2}$); precision = standard deviation of the differences; and LOA = limits of agreement (mean ± 2 standard deviations).

who were free of respiratory disease. P_{etCO_2} bias ranged from small in the respiratory disease-free patients to very large (negative) in patients with pulmonary disease, to extreme in four patients with ARDS (Table 2). Good precision was lacking with both monitoring techniques, as can be seen from the broad limits of agreement shown in Table 2. The figures depicting the limits of agreement between transcutaneous and arterial measurements of P_{CO_2} (Figs. 1A, 2A, & 3A) demonstrate the consistently minimal bias, with a widely varying precision among the different patient groups. Limits of agreement between the P_{etCO_2} vs P_{aCO_2} (Figs. 1B, 2B, & 3B) show the bias, which increases with the severity of underlying respiratory disease, and an unimpressive precision in all patient groups. The frequency distributions of differences for P_{tcCO_2} vs P_{aCO_2} and P_{etCO_2} vs P_{aCO_2} are displayed in Figure 4.

Discussion

Statistical Methodology

Bland and Altman have recommended the analysis of bias and precision as the statistical method of

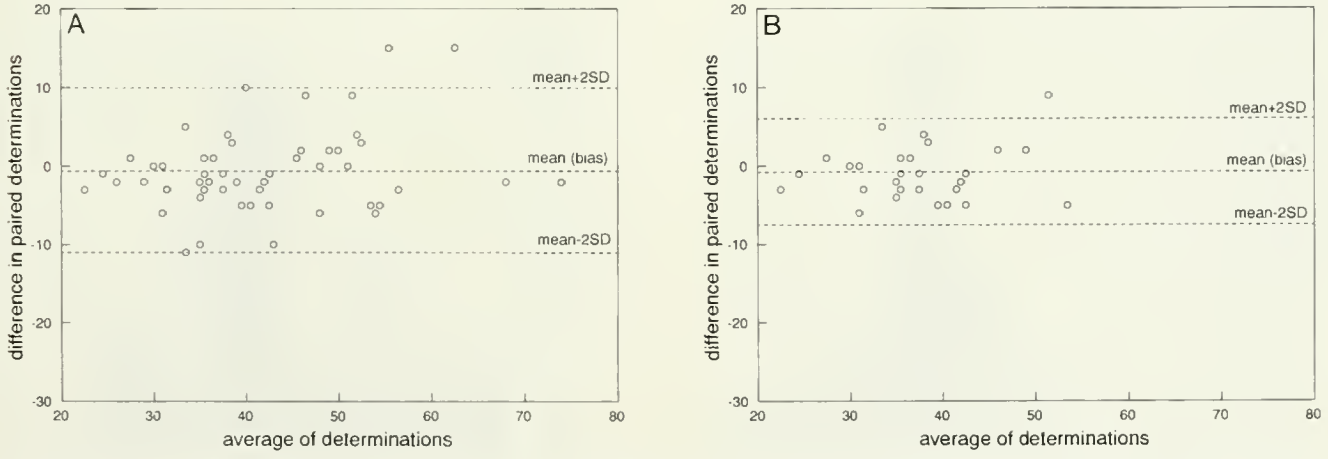

Fig. 1A. Plot of transcutaneous CO_2 and arterial CO_2 differences ($P_{tcCO_2} - P_{aCO_2}$) against mean of $P_{tcCO_2} + P_{aCO_2}$ for all patients. 1B. Plot of end-tidal CO_2 and arterial CO_2 differences ($P_{etCO_2} - P_{aCO_2}$) against mean of $P_{etCO_2} + P_{aCO_2}$ for all patients.

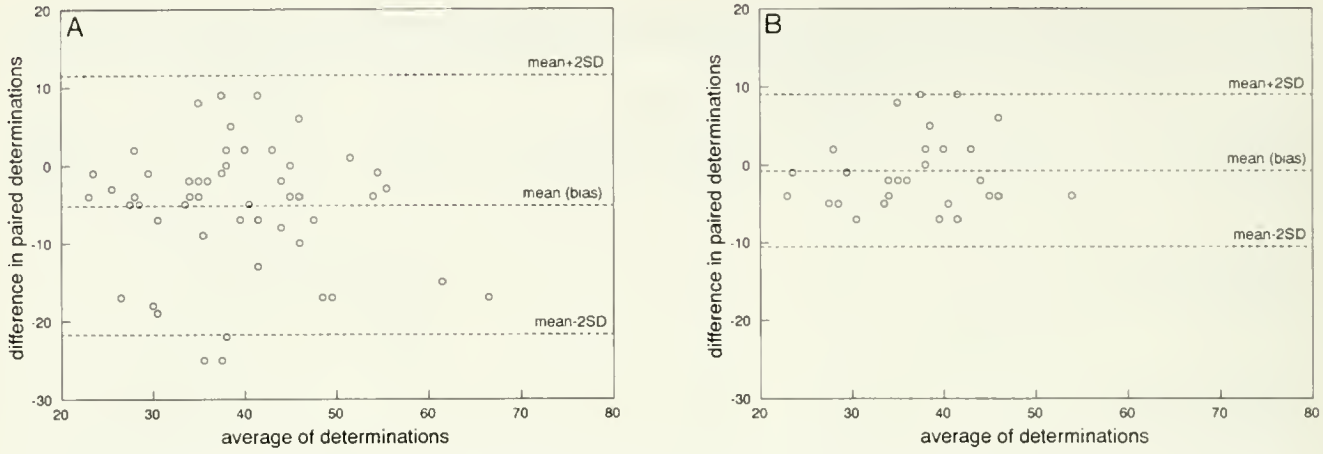

Fig. 2A. Differences against means for transcutaneous CO_2 (P_{tcCO_2}) and arterial CO_2 (P_{aCO_2}) in patients free from respiratory disease. 2B. Differences against means for end-tidal CO_2 (P_{etCO_2}) and arterial CO_2 (P_{aCO_2}) in patients free from respiratory disease.

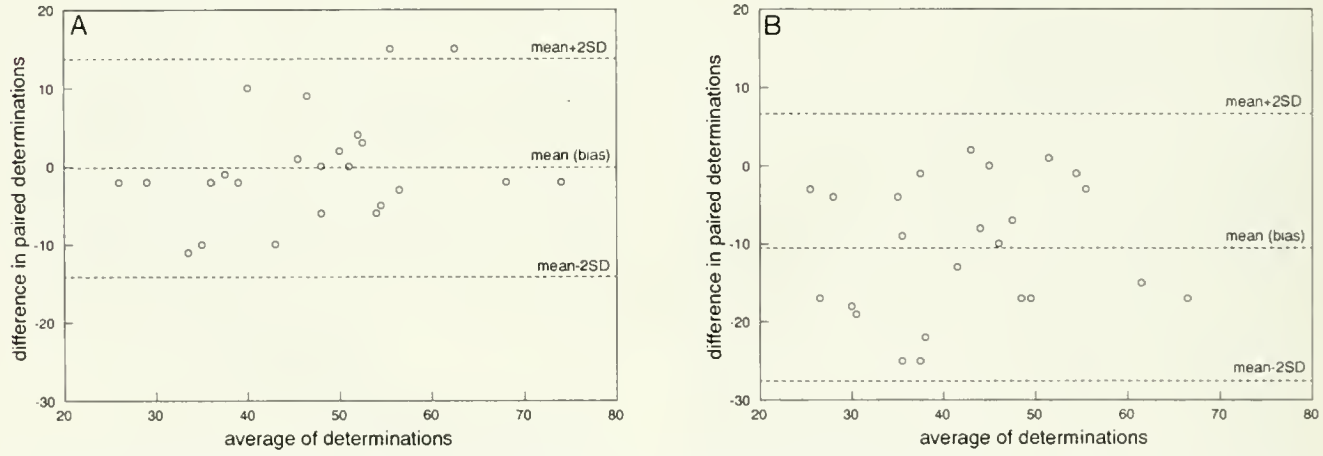

Fig. 3A. Differences against means for transcutaneous CO_2 (P_{tcCO_2}) and arterial CO_2 (P_{aCO_2}) in patients with respiratory disease. 3B. Differences against means for end-tidal CO_2 (P_{etCO_2}) and arterial CO_2 (P_{aCO_2}) in patients with respiratory disease.

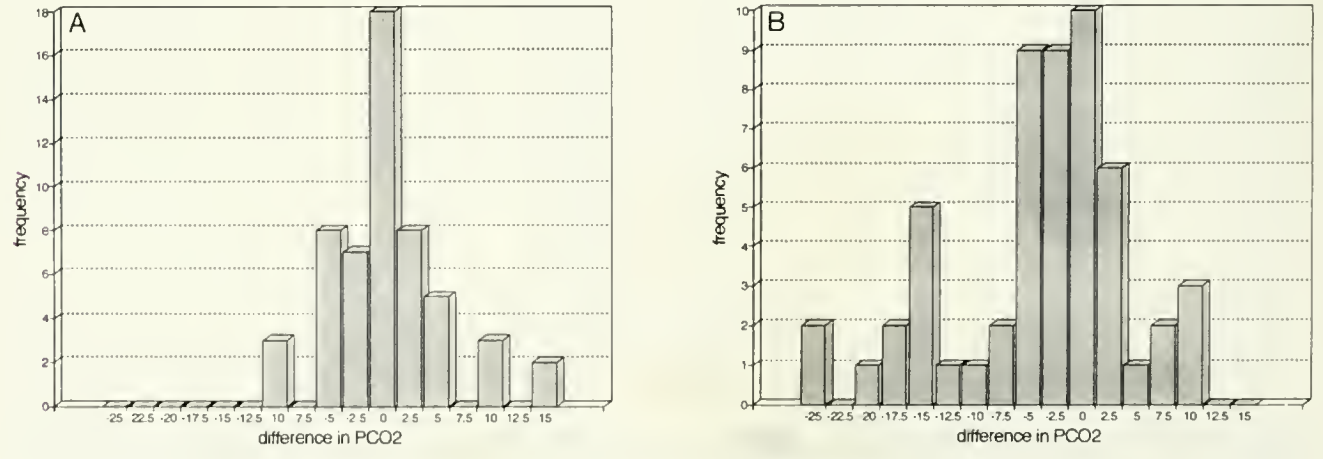

Fig. 4A. Frequency distribution of differences between transcutaneous CO_2 (P_{tcCO_2}) and arterial CO_2 (P_{aCO_2}) ($P_{tcCO_2} - P_{aCO_2}$). 4B. Frequency distribution of differences between end-tidal CO_2 (P_{etCO_2}) and arterial CO_2 (P_{aCO_2}) ($P_{etCO_2} - P_{aCO_2}$).

choice in determining the limits of agreement between two methods of clinical measurement.[19] We took this approach because it has been demonstrated that techniques such as correlation coefficient or regression analysis are not appropriate for assessing measurement agreement. Correlation implies that knowledge of one number allows you to predict the other, but correlation coefficients do not measure agreement, they measure the strength of a relationship.[19] For instance, a transcutaneous monitor that consistently overestimates by 3 torr (bias of +3) and an end-tidal monitor that consistently underestimates by 15 torr (bias of −15) both have a correlation coefficient of "1" signifying a perfect linear relationship.

The ability of a measurement technique to repeatedly result in the same value, under the same conditions, is its precision. In clinical evaluations, some lack of precision is expected because of variability in patient and monitoring conditions, but very imprecise measurements are of little use in clinical situations.

End-Tidal CO_2 Monitoring

End-tidal CO_2 monitors are relatively easy to use, have rapid response times, and provide a continuous estimate of alveolar P_{CO_2}. In normal patients, the difference between P_{etCO_2} and P_{aCO_2} is small. However, P_{etCO_2} may not approximate P_{aCO_2} when $\dot{V}/\dot{Q}$ mismatching or increased dead space is present.[6-8,20-23] Increased dead-space ventilation or ventilation in excess of perfusion can lead to a decrease in exhaled CO_2 because without gas exchange (or with minimal gas exchange) the exhaled CO_2 level approaches that of inspired CO_2, which is near 0. Under these circumstances (high $\dot{V}/\dot{Q}$), CO_2 also may not be adequately removed from the bloodstream and thus arterial CO_2 will increase—further widening the $P_{(a-A)CO_2}$. In lung disease patients, this $\dot{V}/\dot{Q}$ mismatching can be extreme. The CO_2 in the exhaled gas (P_{etCO_2}) can be substantially less than the P_{aCO_2}. In this case, the dead-space-to-tidal-volume ratios (V_D/V_T) are also large and because of this it has been suggested that the P_{etCO_2}-P_{aCO_2} difference [$(P_{(a-A)CO_2})$] correlates closely with V_D/V_T and this P_{etCO_2}-P_{aCO_2} difference may be used to predict the extent of a patient's pulmonary dysfunction and his or her ability to wean.[21]

Besides lung pathology, technical errors with capnography equipment (such as with the analyzer, sampling system, and/or environment changes) can also contribute to the variance between P_{etCO_2} and P_{aCO_2}.[24] This being so, clinicians anticipating close, or constant, correlation between P_{etCO_2} and P_{aCO_2} may be misled, especially in the patient with pulmonary pathology. In patients with pulmonary disease, a fall in P_{etCO_2}, for example, can be misleading because it could be associated with improved $\dot{V}/\dot{Q}$ matching and a greater fall in P_{aCO_2}, no change in $\dot{V}/\dot{Q}$ matching and an equal fall in P_{aCO_2}, or decreased $\dot{V}/\dot{Q}$ matching with an increased P_{aCO_2}.

In our study, variability in $\dot{V}/\dot{Q}$ matching, especially in the respiratory disease group, certainly contributed to the substantial variability in the P_{etCO_2}-P_{aCO_2} relationship and decreased precision.

Transcutaneous CO_2 Monitoring

Transcutaneous monitoring employs a heated electrode with the potential for causing burns (requiring frequent site changes) and has a relatively slow response time, but may be more useful in non-intubated patients than exhaled CO_2 monitoring. When P_{tcCO_2} monitors were first developed, they always overestimated P_{aCO_2} values; consequently, manufacturers compensate for this result of increased skin metabolism due to heating, with temperature correction factors,[11,13,15,16] and thus have improved transcutaneous monitor accuracy but have not eliminated all aberrancies. Transcutaneous electrode temperature was set to 44°C in our study because Tremper et al[25] found that this temperature in adult patients led to a faster response to hypercarbia, shock, and fluid resuscitation. There was little concern for burning these adult patients at this temperature, although manufacturers have recommended the use of a lower temperature, which allows for longer intervals between site changes.

Expanded use of transcutaneous CO_2 monitoring has been recommended. Palisano and Severinghaus have stated that "Transcutaneous CO_2 monitoring deserves far greater use, particularly when end-tidal gas analysis is impractical."[15] Because of the limitations of P_{etCO_2} monitors in $\dot{V}/\dot{Q}$ mismatch, we

postulated that transcutaneous CO_2 would be the technique of choice in patients with pulmonary pathology.

Study Findings

With all data pairs included, determination of limits of agreement revealed that transcutaneous CO_2 monitoring could more accurately predict P_{aCO_2} than could end-tidal CO_2 monitoring in our intensive care patients. However, limits of agreement for both monitoring techniques in predicting P_{aCO_2} demonstrate clinically important errors (Table 2). In two instances, transcutaneous monitoring precision was affected because P_{tcCO_2} values drifted higher while P_{aCO_2} remained unchanged. We have observed this same phenomenon in neonates, which is probably related to increased CO_2 production versus removal at the heated skin site. This causes a rise in P_{tcCO_2} and a widening of P_{tcCO_2}-P_{aCO_2} gradient. Transcutaneous monitor performance in adults was comparable to what we have experienced in neonates. We experienced no technical difficulties due to obesity, and the readings in these patients were of comparable accuracy to patients of normal weight. Our group of patients without documented lung disease demonstrated improved limits of agreement and precision compared to the total group and a nearly identical bias with both monitoring techniques (Table 2). End-tidal CO_2 monitoring offers the benefit of ease of application and may be useful when respiratory disease is not present, although it remained rather imprecise in our group of patients.

With P_{etCO_2} monitoring, the bias was usually large in the context of clinical importance. A fixed bias between techniques could be easily compensated for; however, the variable bias and imprecision of end-tidal CO_2 monitoring limits its usefulness in an inhomogenous variable group of intensive care patients. We did not study the trending abilities of these CO_2 monitors; however, others have. Tremper et al[18] demonstrated the effective trending capabilities of transcutaneous CO_2 monitoring in individual adult patients. Although capnography has become a standard of care because of its ability to noninvasively monitor and trend ventilation during anesthesia, it has been demonstrated that end-tidal CO_2 measurements in the intensive care setting may not accurately trend P_{aCO_2}.[26,27]

We also compared P_{etCO_2} and P_{tcCO_2} to P_{aCO_2} in four ARDS patients. In 9 data sets obtained from these 4 patients, the mean difference (bias) between P_{etCO_2} and P_{aCO_2} was −18.44, demonstrating a dramatic underestimation of P_{aCO_2} by end-tidal CO_2 monitoring. The widened $P_{(a\text{-}A)CO_2}$ in these ARDS patients is in large part due to increased dead space, which may frequently exceed 60% of each breath.[28] High PEEP levels (mean 14 cm H_2O) in this group may have also increased the $P_{(a\text{-}A)CO_2}$.[29] This small group of ARDS patients seems to verify that the size of the $P_{(a\text{-}A)CO_2}$ is related to the magnitude of the V_D/V_T in such patients. The ARDS group was small, and further study of this group is required; however, it appears that ARDS patients who may demand the closest monitoring, offer substantial problems to the clinicians attempting to monitor them noninvasively. If we recognize the imprecision of these noninvasive monitors in patients with moderate-to-severe pulmonary pathology, the fact that transcutaneous monitoring offers dramatically improved bias in the respiratory disease patients makes it the monitor of choice in this patient group.

Surprisingly, one third (10/30) of the P_{etCO_2} vs P_{aCO_2} comparisons in the disease-free group demonstrated a positive bias, with P_{etCO_2} higher than P_{aCO_2}. Our bias in this group was -0.83 ± 4.9 as opposed to the mean difference of -4.6 ± 2.5 as described in patients with normal lungs by Nunn and Hill.[30] The frequently occurring positive bias could be related to the fact that perfusion may be in excess of ventilation immediately after coronary bypass surgery (the majority of our respiratory disease-free group) when the patients are in a horizontal position.[21] These patients are typically ventilated with low respiratory rates, high tidal volumes, and a prolonged expiratory time that should allow more complete emptying of low $\dot{V}/\dot{Q}$ lung units.[21] Administration of high oxygen concentration initially to many of these patients with a consequent Haldane effect has also been reported to produce a positive P_{ECO_2}-P_{aCO_2} difference.[21]

Conclusions

Both end-tidal and transcutaneous CO_2 monitors are imprecise and should be used only as adjuncts to ABG analysis for ventilation monitoring. Monitor refinement or development of a CO_2 monitor

that is less affected by technical errors and physiologic changes would greatly increase the effectiveness of noninvasive ventilatory monitoring. In ventilated patients with respiratory disease, particularly severe lung disease with increased dead space, transcutaneous CO_2 values more accurately reflect arterial P_{CO_2} than do end-tidal CO_2 values. End-tidal CO_2 monitoring generally provides a poor estimate of P_{aCO_2} in ventilated patients with severe respiratory disease. End-tidal CO_2 monitoring and transcutaneous monitoring provide comparable estimates of P_{aCO_2} in ventilated patients without respiratory disease. With current concerns for soaring medical costs, we recommend careful consideration before purchasing end-tidal CO_2 monitoring equipment for critical care areas—especially when transcutaneous monitoring is available.

ACKNOWLEDGMENTS

The authors thank Kathryn Harris for her editorial assistance in the preparation of the manuscript.

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Feasibility of Applying Flow-Synchronized Ventilation to Very Low Birthweight Infants

Gina M Servant BS RRT, Joanne J Nicks RRT, Steven M Donn MD, Kenneth P Bandy BS RRT, Cheryl Lathrop BS RRT, and Ronald E Dechert MS RRT

BACKGROUND: Asynchrony between the very low birthweight (VLBW) baby's spontaneous efforts and ventilator-delivered breaths may result in inefficient gas exchange, pneumothorax, and variability in cerebral blood flow. METHODS & MATERIALS: We evaluated the short-term application of a flow-actuated device (Ventilator Flow Synchronizer, Bird Products Corp, Palm Springs CA) for triggering an otherwise time-cycled, pressure-limited ventilator, in 10 stable VLBW infants. Variables at the end of 2 1-hour periods of conventional intermittent mandatory ventilation were compared to the same variables at the end of 1 hour of flow-synchronized ventilation (FSV). RESULTS: Mechanical rate and minute ventilation increased with FSV. P_{aO_2} rose significantly, but no change was seen in P_{aCO_2}. No significant changes were seen in mean airway pressure or mechanical tidal volume, and no adverse reactions or mechanical malfunctions were observed. CONCLUSION: Short-term evaluation suggests that FSV is safe and feasible in stable VLBW babies. (Respir Care 1992;37:249-253.)

Background

Although intermittent mandatory ventilation (IMV) has been the mainstay of treatment for more than 20 years for newborn infants with respiratory disorders, complications associated with IMV still occur, particularly in the very low birthweight (VLBW) infant. Asynchrony between the baby's spontaneous efforts and ventilator-delivered breaths may result in inefficient gas exchange, pneumothorax, and even variability in the velocity of cerebral blood flow.[1,2] At times these adverse effects may necessitate the use of extreme ventilator settings, sedative agents, and skeletal muscle relaxants.

Various techniques to accomplish patient-triggered ventilation and synchronous breathing have been used.[3-6] Pressure triggering has probably been the most common mode and has been successful in adults and children. Yet, the pressure sensitivity and response time have precluded its use in the VLBW infant. Triggering devices that respond to changes in thoracic impedance are also under investigation.

We evaluated a prototype device that uses changes in gas flow at the proximal airway to trigger the delivery and termination of mechanical breaths. The objective of our short-term pilot study was to determine the feasibility of coupling a flow-synchronized triggering mechanism with a standard system of infant mechanical ventilation without adversely affecting pulmonary gas exchange or mechanics.

Ms Servant and Ms Nicks are Clinical Supervisors in the Respiratory Care Department, Dr Donn is Professor of Pediatrics (Section of Newborn Services), Mr Bandy is Director and Ms Lathrop Assistant Director of the Respiratory Care Department, and Mr Dechert is Director of the Metabolic Laboratory in the Department of Surgery—University of Michigan Medical Center, Ann Arbor, Michigan.

The authors have no financial interest in the product described and received no outside funding for the study.

A version of this paper was presented by Ms Servant at the RESPIRATORY CARE OPEN FORUM during the 1990 AARC Annual Meeting in New Orleans, Louisiana.

Reprints: Gina M Servant BS RRT, Department of Pediatric Respiratory Care, Box 0208, F3064—MCHC, 200 E Hospital Drive, Ann Arbor MI 48109-0208.

Equipment Description

The Ventilator Flow Synchronizer (VFS, Bird Products Corp, Palm Springs CA) was developed to synchronize mechanical ventilator breaths with the infant's spontaneous effort. A sensor placed in the infant's proximal airway detects flow and processes that flow signal to trigger an otherwise time-cycled, pressure-limited ventilator.

The sensor is a variable-area differential-pressure flow transducer. All gas flow into or out of the patient passes through the transducer, resulting in a small pressure difference that is converted to an analog signal. A change in gas flow as small as 0.2 L/min is sufficient to trigger the ventilator. The transducer adds less than 0.5 mL of dead space to the ventilator circuit.

For the present study, the VFS was coupled with a standard infant ventilator, the IV-100B (Sechrist Industries Inc, Anaheim CA). The VFS was programmed to override the internal timing mechanism of the Sechrist ventilator, which continued to provide the gas flow and pressure necessary to deliver mechanical breaths.

The ventilator equipped with the VFS initiates a breath under two conditions. The first is detection of inspiratory effort; the second is passage of the period of time set by the breath-rate control. In the absence of spontaneous effort, the ventilator continues to deliver mandatory breaths. Similarly, mechanical breaths are terminated under two conditions. The first is detection of an expiratory trigger event (eg, when the expiratory trigger is set at P-25, inspiration is automatically terminated when inspiratory flow drops to 25% of peak flow). The second is the passage of the period of time determined by the inspiratory time-limit control.

Methods of Evaluation

We evaluated the short-term application of the VFS in a convenience sample of 10 preterm infants whose gestational ages ranged from 24-31 weeks and whose weights at the time of the study ranged from 480-1,400 g (Table 1). All infants were in the convalescent phase of respiratory distress syndrome characterized by a stable ventilator course prior to the study. All patients had indwelling arterial catheters for blood gas analysis and pressure determinations. The study was approved by the Institutional Review Board of the University of Michigan Medical Center. Patients were evaluated during conventional mechanical ventilation by standard cardiorespiratory monitoring. Pressure-volume and flow-volume measurements were detected and recorded by the Star Calc System (Infrasonics Inc, San Diego CA). Calibration of the device was done prior to testing according to the specifications of the manufacturer. Arterial blood gases were measured by an automated blood gas analyzer.

Table 1. Description of Patients Entered into Flow-Synchronized Ventilation Study

Patient	Gestational Age (wk)	Weight (g)	Actual Age (days)	Days on Ventilator*
1	30	610	4	4
2	29	1,040	6	6
3	26	720	2	2
4	27	1,200	1	1
5	27	860	1	1
6	24	480	7	7
7	28	880	3	3
9	31	1,400	1	1
10	31	1,240	8	8

*prior to study.

Each patient was monitored continuously during the 3 hours of the study by cardiorespiratory monitor, transcutaneous P_{CO_2}-P_{O_2} monitor, and pulse oximeter. At the end of the first hour of conventional ventilation (IMV_1), measurements including vital signs, arterial blood gases, pulse oximetry, mean airway pressure ($\bar{P}_{aw}$), inspiratory time (t_I), and pulmonary mechanics were made. Patients were switched to the Flow Synchronizer (FSV) for 1 hour and measurements were repeated at the end of the hour. The patients then received an additional hour of conventional ventilation (IMV_2) with final measurements obtained at the end of the study period. No changes in ventilator settings were made, including PIP and PEEP, during the entire 3-hour study period. Inspiratory time on the VFS was set at P-25 for all patients, enabling them to control their own inspiratory time. The maximum inspiratory time on the VFS was set to match the inspir-

Table 2. Comparison of Measured Variables in 10 Premature Infants during Three Successive 1-Hour Study Periods

Variable	IMV_1* mean (SD)	FSV† mean (SD)	IMV_2 mean (SD)	p
Mechanical rate breaths/min	27 (6.5)	54 (25.8)	27 (6.4)	0.0014
$\bar{P}_{aw}$ cm H_2O kPa	6.3 (1.4) 0.6 (0.1)	7.3 (1.2) 0.7 (0.1)	6.3 (1.3) 0.6 (0.1)	NS
Inspiratory time seconds	0.36 (0.09)	0.26 (0.09)	0.36 (0.04)	0.0278
V_T mL	9.1 (3.1)	8.7 (3.7)	9.5 (2.5)	NS
$\dot{V}_E$ mL	366.5 (140) mechanical + spontaneous	521.8 (119.8)	394.5 (150.0) mechanical + spontaneous	0.016 0.143 IMV to FSV
P_{aO_2} torr kPa	68 (7.3) 9.1 (1.0)	84 (12.5) 11.1 (1.7)	79 (10.9) 10.5 (1.5)	0.0385
P_{aCO_2} torr kPa	37 (7.9) 4.9 (1.1)	40 (9.3) 5.3 (1.2)	36 (6.9) 4.8 (0.9)	NS

*IMV = intermittent mandatory ventilation.
†FSV = flow-synchronized ventilation.

atory time set on the Sechrist during IMV_1 and IMV_2.

Measured variables expressed as the mean (SD) were analyzed by repeated measures analysis of variance using Hotelling's t test.[7] Significance was considered to be $p < 0.05$.

Results of Evaluation

Table 2 lists the mean (SD) for variables measured during the three study periods. Mechanical respiratory rate and total minute ventilation showed a statistically significant increase during FSV compared both to IMV_1 and IMV_2. No significant changes occurred in P_{aCO_2}. We did note a significant rise in P_{aO_2} from IMV_1 to FSV. This effect appeared to be sustained during IMV_2.

Inspiratory time showed a significant decrease during FSV compared to IMV_1. Differences in inspiratory time between FSV and IMV_2 showed a trend that did not reach significance. This may have been due to a slight variation in inspiratory time during IMV_2 for 3 of the 10 patients.

No significant changes were noted for $\bar{P}_{aw}$ or mechanical V_T during any of the study periods, and no adverse reactions or mechanical malfunctions were observed in any of the study patients.

All 10 patients demonstrated synchronized breathing in FSV when compared to IMV. Figure 1 compares the flow-volume loops between IMV_1 and FSV in 4 different patients. Note the asynchrony versus synchrony of breathing when the two modes of ventilation are compared.

Discussion

The observed increases in mechanical respiratory rate and total minute ventilation (mechanical + spontaneous, when in IMV) represent the additive effects of patient triggering during FSV. The increased minute ventilation was associated with a concomitant rise in P_{aO_2}; however, no associated decrease in P_{aCO_2} was observed. The reason for this discrepancy remains unclear. We can only speculate about why CO_2 levels did not change sig-

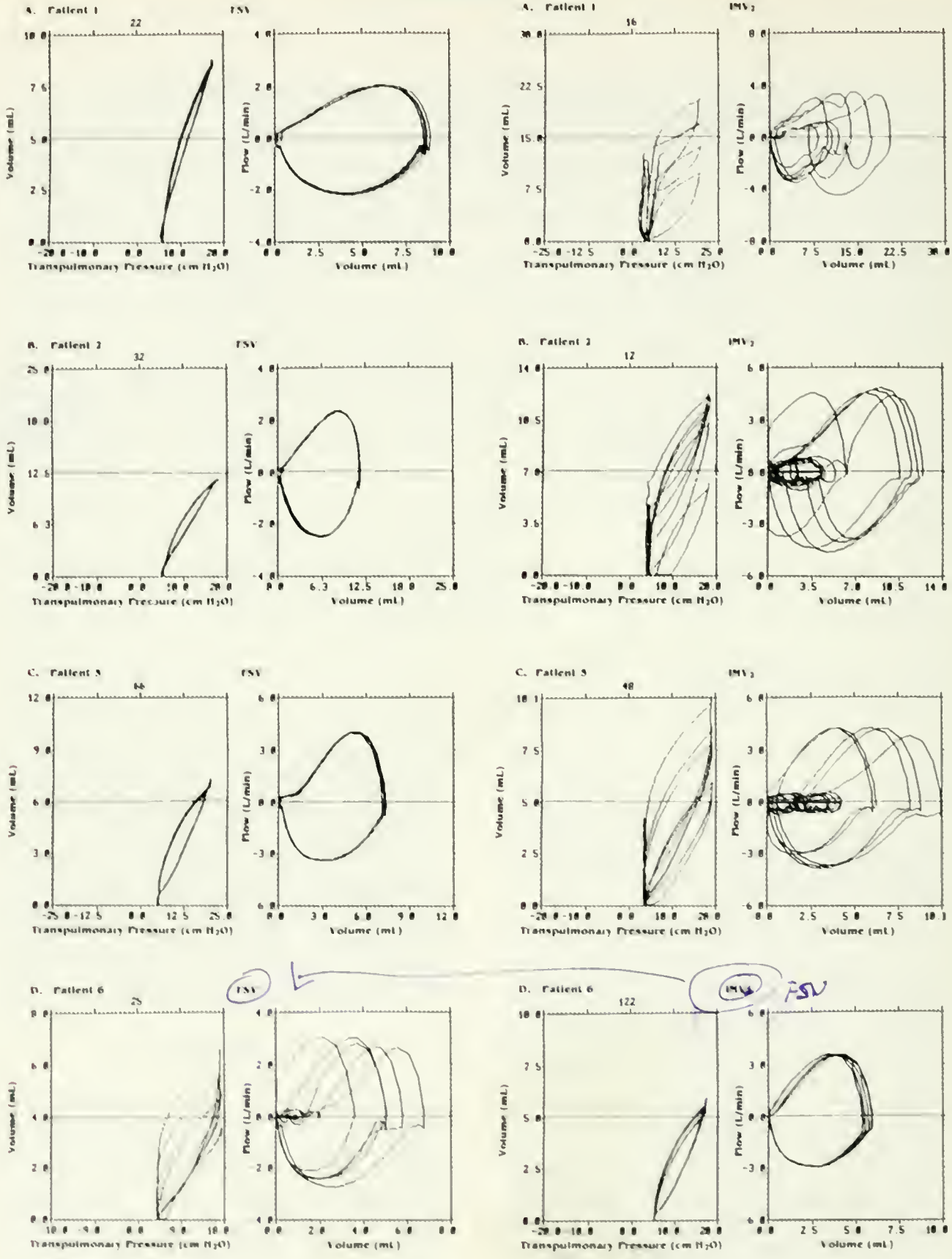

Fig. 1. Flow-volume and pressure-volume loops for Patients 1 (A), 2 (B), 5 (C), and 6 (D) demonstrating asynchronous breathing during IMV_1 or IMV_2 and synchronous breathing during FSV.

nificantly despite a significant increase in total minute ventilation during FSV. Greenough et al reported similar results of increased oxygenation and minute ventilation during patient-triggered ventilation with no decrease in $P_{a(c)_2}$.[5] Evaluation of this physiologic phenomenon has not been studied and needs to be evaluated in a larger controlled, randomized clinical trial. The short inspiratory time observed in the study patients during FSV was less than the t_i usually chosen in our institution during conventional mechanical ventilation. In FSV, the termination of the inspiratory cycle is determined by the reduction of inspiratory flow to 25% of peak flow when expiratory trigger is set at P-25. Therefore, the decreased compliance and increased resistance of a premature lung with RDS most likely influenced the inspiratory time during FSV as compared to IMV during which the inspiratory time was held constant.

Mitchell and colleagues[6] have reported their experience with a similar system of patient-triggered ventilation. However, they concluded that long-term patient-triggered ventilation was impractical for infants less than 28-weeks gestation. Predictors of failure with their device included a lack of improvement in oxygenation and a somewhat slow triggering rate at 1 hour. The design of their device differs from the VFS in that mandatory breaths are delivered only when the sensor fails to detect respiratory effort for 8 seconds. Greenough et al[5] have suggested that a more effective means of flow-sensitive patient triggering would include a minimum number of predetermined ventilator breaths delivered automatically in the absence of patient effort. The VFS has that capability, up to IMV rates of 80/min.

This short-term evaluation of the VFS in 10 stable, mechanically ventilated preterm infants recovering from RDS suggests that the device is safe and its use feasible in VLBW babies. An infant weighing 480 g was capable of triggering mechanical breaths and synchronizing respiratory effort with this system, with no evidence of fatigue. A long-term study looking at more specific outcome parameters is underway to evaluate synchronous breathing as a better alternative to routine IMV and to better delineate the physiologic changes that occur with synchronous assisted mechanical ventilation in premature infants.

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Reactive Airway Dysfunction Syndrome (RADS): A Report of Three Cases

Vito A Angelillo MD

Background

Exposure to various dusts, chemicals, and gases, either in the work place or in the home setting, is known to cause acute respiratory symptoms.[1-4] This is usually due to the irritant nature of such agents and is primarily manifested by cough. On occasion, chest tightness, wheezing, and dyspnea resembling an acute asthmatic attack occur. Such episodes are usually short-lived and respond to symptomatic management with bronchodilators. Many patients have no further difficulty or only experience symptoms on re-exposure to the offending agent. However, some have developed clinical asthma after repeated exposure and are said to have occupational asthma.[1,2]

The reactive airway dysfunction syndrome (RADS) described by Brooks et al[5] in 1985 is characterized by the appearance of asthma-like symptoms and an increase in nonspecific bronchial responsiveness after a single, acute, massive exposure to irritants. Until the report of Brooks et al, it was generally held that single exposures to irritant dust or fumes did not result in prolonged impairment of pulmonary function. However, as the three cases presented illustrate, patients may go undiagnosed and inadequately treated. They may also be labeled as chronic complainers or be motivated by the possibility of financial compensation.

Dr Angelillo is associated with the Pulmonary Division, Department of Medicine, Creighton University School of Medicine, Omaha, Nebraska.

Reprints: Vito A Angelillo MD, Pulmonary Division, St Joseph Hospital, Suite 3820, 601 North 30th Street, Omaha NE 68131.

Case Reports

The three patients were employees of a beef-processing plant. On the evening of July 17, 1986, a cleanup crew was working with a solution of phosphoric acid and a chlorinated disinfectant. Ordinarily, each agent is used separately to clean organic debris from equipment. For reasons unknown, the agents were mixed in a container holding 40 gallons of water. A visible yellow fog with a strong odor of chlorine was reported to have risen immediately from the container. Chlorine levels were not available.

Chlorine gas is an irritant that affects mucous membranes of the eyes and upper and lower respiratory tract, causing conjunctivitis, laryngitis, bronchitis, and, occasionally, pulmonary edema. NIOSH has recommended a ceiling limit of 0.5 ppm for a 15-minute sampling period.[6]

All patients were evaluated and treated initially and over the subsequent 4-year period by their primary care physicians. Spirometric studies were done either in the office or at local hospitals. Methacholine challenges were performed by standard technique[7] (4 years after the acute exposure) when the three patients were referred to the same pulmonologist because of persistent respiratory symptoms and pending litigation.

Case 1

The patient was a 26-year-old White man who immediately following exposure was brought to the emergency room complaining of burning eyes, sore throat, and cough. A chest x-ray was normal and arterial blood gas values on oxygen at 5 L/min via nasal cannula were P_{O_2} 172 torr, P_{CO_2} 36 torr, and

pH 7.46. Spirometry performed the following morning revealed forced vital capacity (FVC) 5.49 L (105% of predicted), forced expiratory volume in the first second (FEV_1) 4.58 L (104% of predicted), FEV_1/FVC 83%, and forced expiratory flow ($FEF_{25-75\%}$) 4.49 L/s (95% of predicted). Prior to the incident, the patient had been in good health, having been a collegiate wrestler. He was a nonsmoker and had no family history of any pulmonary disease.

During the ensuing 4 years, he had frequent office visits for chronic cough with sputum production and dyspnea on exertion, which worsened with exposure to cold air. He was treated primarily with antibiotics. Spirometry performed on 12-1-87 revealed FVC 5.16 L (96% of predicted), FEV_1 4.74 L (106% of predicted), FEV_1/FVC 92%, and $FEF_{25-75\%}$ 5.96 L/s (124% of predicted). On 11-22-90, spirometry revealed FVC 5.52 L (104% of predicted), FEV_1 4.56 L (107% of predicted), FEV_1/FVC 83%, and $FEF_{25-75\%}$ 4.54 L/s (101% of predicted). A methacholine inhalation challenge was markedly positive at a PC_{20} of 0.67 mg/mL. He was treated with oral theophylline and an albuterol inhaler with symptomatic relief.

Case 2

The patient was a 28-year-old White man, who after observing the spread of gas fumes spent almost 20 minutes making sure that the area was evacuated. He complained of a severe sore throat, burning eyes, and dyspnea. In the emergency room, his chest x-ray was normal and spirometry revealed FVC 6.11 L (114% of predicted), FEV_1 4.91 L (113% of predicted), and FEV_1/FVC 80%. No wheezing was present on auscultation.

The patient was a nonsmoker with no family history of lung disease. During the ensuing 4 years, he had numerous outpatient visits for complaints of productive cough, intermittent sore throat, hoarseness, fatigue, dyspnea, and wheezing, which worsened with exposure to cold and cigarette smoke. He was treated for bronchitis primarily with antibiotics. On one occasion, he had been treated with theophylline, prednisone, and a beclomethasone inhaler with good relief, but this treatment was not continued. Spirometry on 8-14-86 revealed FVC 5.18 L (83% of predicted), FEV_1 4.22 L (82% of predicted), and FEV_1/FVC 82%. Studies performed on 1-25-90 revealed FVC 4.97 L (93% of predicted), FEV_1 3.99 L (96% of predicted), FEV_1/FVC 80%, and $FEF_{25-75\%}$ 3.61 L/s (83% of predicted). A diffusion study revealed a diffusing capacity of 97% of predicted. A methacholine challenge was markedly positive at PC_{20} of 1.8 mg/mL. He was treated with oral theophylline and albuterol and beclomethasone via inhaler with good control of his symptoms.

Case 3

The patient was a 25-year-old White man, a supervisor, who was operating a forklift when the accident occurred. He continued to work for 4 to 5 minutes in the yellow-green haze before leaving because of burning eyes, cough, and dyspnea. In the emergency room, his chest x-ray was normal, and oxygen saturation was 96% by oximetry on room air. FVC was 4.88 L (97% of predicted), FEV_1 4.12 L (97% of predicted), FEV_1/FVC 85%, and $FEF_{25-75\%}$ 4.30 L/s (92% of predicted). Prior to the exposure he had been in good health with a negative family history. He had been a cigarette smoker of two packs per day but quit at the time of the accident.

Over the next 4 years, he continued to complain of dyspnea and productive cough and was treated primarily with antibiotics. On several occasions he was treated with theophylline with some improvement, but the theophylline was not continued. On 9-30-88, spirometry revealed FVC 5.07 L (105% of predicted), FEV_1 4.20 L (106% of predicted), FEV_1/FVC 81%, and $FEF_{25-75\%}$ 5.29 L/s (97% of predicted). A diffusion study revealed a diffusion capacity of 124% of predicted. Studies were performed again on 1-25-90, with FVC 5.20 L (108% of predicted), FEV_1 4.37 (112% of predicted), FEV_1/FVC 84%, and $FEF_{25-75\%}$ 4.90 L/s (113% of predicted). A methacholine challenge was markedly positive at PC_{20} of 0.73 mg/mL. He was treated with albuterol and cromolyn sodium via inhaler with good results.

Discussion

Since Brook et al's[5] first description of the reactive airway dysfunction syndrome, surprisingly few

reports have appeared in the literature.[8-10] This most likely accounts for the fact that few physicians appear to be aware that asthma as a long-term sequela can occur after a single exposure to an irritant substance. This lack of awareness is further supported by the extended interval between time of the incident and the actual diagnosis, which has ranged from 2 to 72 months.[5,8-10] Signs and symptoms in the three cases reported persisted for 4 years before diagnosis.

Table 1 lists the criteria for the diagnosis of RADS.[5] It is important to emphasize that a methacholine challenge may be necessary to confirm the presence of increased airway reactivity because it is not unusual to find normal pulmonary function tests despite persistent symptoms. It is further important to point out that RADS differs from typical occupational asthma. Occupational asthma develops after a period of sensitization.[1,2] In occupational asthma, the diagnosis of the respiratory problems is made easier because of the association with known and repeated exposure to a particular agent, and recovery is possible if there is early withdrawal from the offending agent, although long-term disease can persist even after withdrawal.[10] The natural history and prognosis of RADS has yet to be determined.

Table 1. Criteria for the Diagnosis of Reactive Airway Dysfunction Syndrome (RADS)

A documented absence of preceding respiratory complaints
Onset of symptoms after a single specific exposure incident or accident
Exposure to a gas, smoke, fume, or vapor present in very high concentrations, with irritant qualities
Onset of symptoms within 24 hours after exposure that persists for at least 3 months
Symptoms that simulate asthma with cough, wheezing, and dyspnea predominating
Pulmonary spirometric tests may or may not suggest airflow obstruction
Positive response to methacholine challenge
Absence of other types of pulmonary disease

The exact pathologic change induced after inhalation injuries is not known, but bronchiolitis obliterans has been proposed as a lesion occurring after inhalation of some toxic gases and fumes.[11] In two cases reported by Brooks et al[5] bronchial biopsies were available. Respiratory epithelial injury with a chronic nonspecific inflammatory response was present without evidence of bronchiolitis obliterans. Bronchiolitis obliterans is a fibrotic lung disease that can develop after toxic fume inhalation, and usually occurs 2 to 4 weeks after the patient has recovered from an episode of pulmonary edema (ARDS) secondary to the inhalation event.[12,13] At the time the bronchiolitis obliterans develops, the chest x-ray usually shows a reticulonodular pattern, and pulmonary function tests reveal an irreversible restrictive and/or obstructive pattern.[14] Whether bronchiolitis obliterans is present in some patients with RADS is unknown, but the clinical and physiologic courses of the cases presented here and in previous reports are inconsistent with that entity.

It is believed that the bronchial hyperreactivity begins with the loss of respiratory tract epithelium that occurs as a result of the initial inhalation injury.[15,16] Various animal models of respiratory epithelial injury have shown that a number of mechanisms may cause or potentiate bronchial hyperreactivity.[17] These include sensitization of rapidly adapting vagal sensory afferent nerve endings, prevention of the protective release of large concentrations of prostaglandin E_2, generation of 15-lipoxy-genase pathway products, and the deficiency or absence of epithelium-derived "relaxant factor."[18,19]

The exact incidence of nonspecific bronchial hyperreactivity in the general population is unknown, but the estimate is 3 to 19% of asymptomatic normal subjects.[20,21] Because no testing was available prior to the accident, the baseline status of these three patients is unknown. The patient in Case 3 was a cigarette smoker up until the time of the accident. Increased nonspecific bronchial reactivity has been noted in some cigarette smokers.[22] However, our three patients were asymptomatic at the time of exposure and had no history to suggest atopy either in themselves or their families. It is difficult to accept the development of bronchial hyperreactivity in three individuals at the same time as a fortuitous event.

Although it is important for physicians practicing occupational, industrial, or pulmonary medicine to be aware of this syndrome, it is probably just as important, if not more so, for those in pri-

mary care because they will be among the first to be sought out for diagnosis and treatment. There is a tendency to dismiss chronic complaints that are ascribed to a single event; so, it is important to keep RADS in mind when evaluating patients with asthma symptoms and consider a methacholine challenge to confirm the presence of reactive airways.

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An Approach to Metabolic Acidosis for the Respiratory Care Practitioner

Richard E Neiberger RRT MD PhD

Introduction

Conditions producing metabolic acidosis are common and extremely important, complicating conditions in many acutely ill patients cared for by the respiratory care practitioner. In addition, these conditions are frequently difficult to understand and receive the least detailed treatment in our current respiratory therapy textbooks. When I first began in respiratory therapy in 1967, I found the metabolic acidoses the most difficult topic to organize coherently. During the last 25 years, I have developed a strategy that, in algorithmic form, provides a logical approach to evaluation of metabolic acidosis. This approach presupposes a knowledge of normal acid-base physiology. In this paper, I describe an approach to the evaluation of metabolic acidoses. It is my hope that this approach will lead to increased understanding by other respiratory care practitioners providing care, particularly in intensive care units, for patients with pure or mixed metabolic acidoses. It is also my hope that increased understanding of this complex group of conditions will ultimately lead to improvement in the care provided to these patients.

Metabolic acidosis is characterized by a low arterial pH (increased hydrogen ion concentration) and a reduced plasma bicarbonate concentration. Under normal circumstances, the excess hydrogen ion concentration would be expected to increase ventilation, resulting in a decreased P_{aCO_2}. In patients with abnormal lung function, the compensatory respiratory response to metabolic acidosis is incomplete or delayed.

Dr Neiberger is Assistant Professor, Division of Pediatric Nephrology, University of Florida College of Medicine, Gainesville, Florida.

Metabolic acidosis is produced by four basic processes: (1) decreased production of buffer (usually HCO_3^-), (2) increased loss of buffer, (3) increased load of hydrogen ions (may be endogenous or exogenous), or (4) decreased ability to excrete hydrogen ions.

The pH of a solution is the negative logarithm of the hydrogen ion concentration. My personal feeling is that continuing to use the pH system tends to make understanding acid-base problems more difficult. The pH system was developed at a time when multiplication and division by small numbers (ie, 10^{-9} mols, or nanomols) was difficult, and the use of logarithms greatly facilitated mathematical calculations. Now, with calculators and computers, the aid to calculation provided by logarithms is no longer of benefit. We should deal with disorders of hydrogen ion concentration in a manner similar to the way we deal with disorders of any other ion (eg, Na^+, K^+, Ca^{++}). I hope that some day we can do away with the archaic pH system in clinical medicine. For now, we are 'stuck' with it.

The relationship between $[H^+]$ and pH is

pH	7.0	7.1	7.2	7.3	7.4	7.5	7.6	7.7
$[H^+]$ nmol/L	100	79	63	50	40	32	25	20

Approach to Metabolic Acidosis

First establish the presence of metabolic acidosis (Fig. 1). When evaluating the status of an individual, it is extremely important to consider both the clinical presentation and the laboratory data. Discussion with the patient's physician and nurse, along with review of the medical record to obtain a careful history, can simplify even the most complex set of blood gas and electrolyte data. The number and complexity of conditions producing metabolic

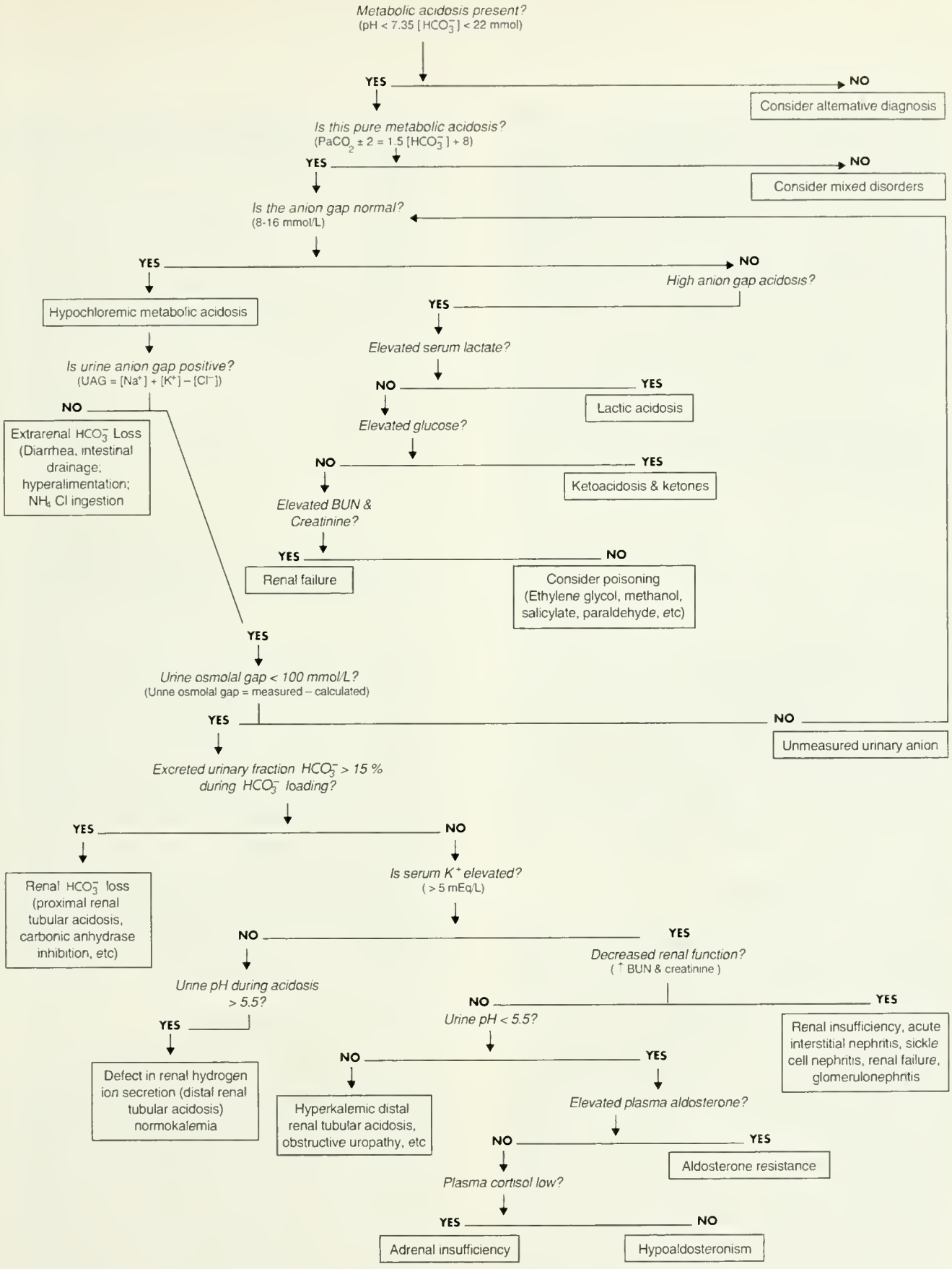

Fig. 1. Algorithm for determining etiology of metabolic acidosis.

acidosis make analysis by looking only at the pH, P_{aCO_2}, and HCO_3^- very superficial.

Metabolic acidosis is present when the serum pH is less than 7.35 and the serum bicarbonate less than 22 mmol/L. If either of these two conditions is not met, then the patient has a mixed acid-base abnormality—or it is unlikely that metabolic acidosis is the primary problem, and an alternative explanation should be sought.

Simple vs Mixed Metabolic Acidosis

In simple metabolic acidosis, the expected respiratory response is hyperventilation. The expected steady-state response of full compensation is predicted by the equation

$$P_{aCO_2} \pm 2 = 1.5\,[HCO_3^-] + 8.$$

If this expected decrease in P_{aCO_2} is not found, then either the patient is not in steady-state compensation, or a mixed acid-base disturbance is present. Delay in progression to steady state is common in patients with lung disease. Reduction of P_{aCO_2} by increased ventilation is the mechanism by which compensation occurs. Reduced lung function may markedly delay the hyperventilation necessary to move to steady-state compensation. In metabolic acidosis, determination of the anion gap is the next step in evaluation (Fig. 1).

Mixed Acid-Base States

In mixed acid-base states, two or more acid-base states occur together. Mixed disorders occurring with metabolic acidosis include (1) metabolic acidosis with respiratory acidosis (in this case, the bicarbonate is less than normal and the P_{aCO_2} is greater than normal); (2) metabolic acidosis with respiratory alkalosis (the primary disease produces a lower than normal P_{aCO_2}—an example would be an individual hospitalized for acute renal failure who develops gram-negative sepsis, producing hyperventilation); and (3) metabolic acidosis combined with metabolic alkalosis (in this combination, blood gas values have no specific pattern—an example would be pancreatitis requiring gastric suctioning occurring in a patient with end-stage renal disease on maintenance hemodialysis). In the metabolic acidosis-alkalosis example, renal failure leads to accumulation of acid metabolic products, whereas suctioning of acid gastric material leads to alkalosis.

The clinical manifestations of acid-base disorders are so nonspecific that evaluation for suspected acid-base disorders is heavily dependent on laboratory assessment to develop an accurate database. In the evaluation of mixed acid-base states, I cannot overemphasize the importance of considering the history, physical examination, and previously measured serum and urine electrolytes. Mixed acid-base disorders are impossible to distinguish on the basis of blood-gas values alone.

The Serum Anion Gap

After the presence of metabolic acidosis has been established, calculate the serum anion gap or unmeasured anion (Fig. 1). These terms are misnomers. There is no real anion gap. The number of cations in serum is equal to the number of anions. The term "unmeasured anion" is inaccurate. There are more than 8-16 mmol/L unmeasured anions in serum. The anion gap is calculated as $Na^+ - (Cl^- + HCO_3^-)$ or $(Na^+ + K^+) - (Cl^- + HCO_3^-)$. Serum potassium is 3 to 6 mEq/L. It is usually easiest to omit potassium from the calculation and use the first of the two equations. The serum anion gap is normally 8-16 mmol/L.

The importance of the anion gap is simply to narrow the diagnostic possibilities. As shown in the algorithm, going down one of the two major pathways is simpler than considering all possible conditions causing metabolic acidosis.

Elevated Anion Gap Acidosis

If the anion gap is widened, then some not routinely measured substance is providing an anion plus free protons. The anion gap is a measure of the unmeasured anions in serum, from which the unmeasured cations have been subtracted (unmeasured anion – unmeasured cations = anion gap). It is important to exclude two uncommon possibilities first: (1) the widened anion gap is caused by decreased unmeasured cations (hypokalemia, hypocalcemia, hypomagnesemia) or (2) laboratory error (falsely elevated sodium or falsely decreased chlo-

ride or bicarbonate). Once these have been excluded, a differential diagnosis can be organized (Fig. 1 or Table 1). The acid may be produced endogenously (ie, lactic acid), in excess quantities or in normal amounts in the presence of decreased renal excretion (endogenous). The acid substance may be ingested or administered (exogenous).

Table 1. Common Causes of Metabolic Acidoses Associated with Increased Anion Gap

Endogenous causes
Lactic acidosis
Renal failure
Ketoacidosis
Massive cellular destruction (ie, rhabdomyolysis or tumor lysis syndrome)
Organic acidemia
Exogenous causes
Ingestion or administration of salicylate, methanol, formaldehyde, ethylene glycol, paraldehyde, sulfate, toluene, and many others

The concentration of many endogenous acid-producing substances can be measured directly (Fig. 1). However, recognition of exogenous chemicals usually requires historical information. Sometimes these chemicals can be recognized by using a metabolic screen. Renal insufficiency is recognized in a patient with oliguria, rising serum creatinine, and/or blood urea nitrogen (BUN).

In a setting in which the presence of an exogenous low-molecular weight substance such as methanol, ethanol, ethylene glycol, or mannitol is suspected, it may be helpful to measure the serum osmolar gap. The osmolarity of serum can be measured directly. In addition, the serum osmolality can be estimated:

$$\text{Estimated serum mOsm} = 2[Na^+ + K^+] + [\text{urea}] + [\text{glucose}].$$

The concentrations of serum sodium, potassium, urea, and glucose must be expressed in mmol/L. When the measured serum osmolality exceeds the calculated osmolality by more than 10 mOsm/kg water, a search for an unidentified osmole should be undertaken.

Normal Anion Gap Acidosis

Metabolic acidosis in combination with a normal anion gap (8-16 mmol/L) leads to the following diagnostic possibilities (Table 2): (1) loss of bicarbonate from the gastrointestinal tract (many mechanisms are possible, including diarrhea, fistulae, and urinary diversion), (2) renal diseases producing decreased hydrogen ion excretion or decreased bicarbonate reabsorption, (3) dilutional acidosis, (4) administration of chloride-containing acid solutions, (5) administration of ion-exchange resins (eg, cholestyramine), (6) recovery phase of either respiratory alkalosis or mild ketoacidosis, and (7) acidosis produced by movement of intracellular hydrogen ions to the extracellular space.

Table 2. Common Metabolic Acidoses Associated with Normal Anion Gap (Hyperchloremic Metabolic Acidosis)

Gastrointestinal HCO_3^- loss
Renal HCO_3^- loss
Decreased renal H^+ ion excretion
HCl or other cation-chloride substance
Endogenous
Exogenous

Starting with hyperchloremic metabolic acidosis (Fig. 1), review the overall evaluation of normal anion gap acidosis. When the bicarbonate is low, the most common other anion to increase is chloride. These acidoses are frequently referred to as hyperchloremic metabolic acidosis.

Urine Anion Gap

In patients with hyperchloremic metabolic acidosis, calculating the urine anion gap is the most helpful next step (Fig. 1). Depending on the urine anion gap, one is directed down a pathway evaluating either renal causes of acidosis or non-renal mechanisms producing acidosis.

The urine anion gap is calculated as urinary $[Na^+ + K^+] - Cl^-$. If the urine chloride is greater than $Na^+ + K^+$, then either (1) urinary ammonium (NH_4^+) is more than 80 mmol/day (a response that should prevent acidosis), or (2) some unmeasured urinary anion is present.

A simple test to rule out the presence of another anion is to compare the measured and calculated urine osmolality. The urine osmolality is calculated as $2[Na^+ + K^+] + [\text{urea}] + [\text{glucose}]$. These substances must be expressed as mmol/L. A small urine sample can be sent to the laboratory for meas-

urement of urine osmolality. The urine osmolar gap is calculated as measured urine osmoles minus calculated urine osmoles. If the urine osmolar gap is greater than 100 mOsm/L, an unaccounted for anion in the urine should be suspected. If this is the case, recheck the serum anion gap and serum osmolar gap for an unmeasured serum anion (Fig. 1). If an unmeasured anion is present, it should be in both serum and urine. When results are inconsistent, always be sure to consider the possibility that there is an error in measurement or calculation of serum anion gap, serum osmolar gap, urine anion gap, or urine osmolar gap.

If the urine anion gap is positive and no evidence for an unmeasured anion can be found, then the ammonium production is probably normal. Urine ammonium can be measured directly; however, it is not a routine laboratory test at most hospitals. If the rate of ammonium (NH_4^+) excretion is normal (negative urine anion gap), the kidney is not responsible for the acidosis. Thus, investigation should be directed at identifying a non-renal cause of metabolic acidosis (Fig. 1). If the urine anion gap is negative, a renal mechanism is a likely cause for the metabolic acidosis.

Up to this point, all the evaluation has involved consideration of routinely performed tests such as arterial blood gas analysis, measurement of serum and urine electrolytes, and measurement of serum and urine osmotic pressures. The remainder of the evaluation is directed at identifying a specific renal mechanism as a source for metabolic acidosis. There are several possible mechanisms, but, in simplest terms, the kidney does not reabsorb bicarbonate or secrete acid, or produce ammonium properly, or some combination of these. Any of these problems can be caused by an intrinsic renal abnormality or by the kidney as an end-organ (ie, carbonic anhydrase inhibition or hypoaldosteronism).

Low proximal tubular bicarbonate reabsorption may be caused by a number of drugs in addition to having acquired and genetic causes (Table 3). If, by history, one or more of these conditions are present, there may be no need to proceed further. On the other hand, if no previously recognized association for the production of metabolic acidosis can be found, then in cooperation with the patient's physician, the fractional bicarbonate excretion should be measured.

Table 3. Proximal Renal Tubular Acidosis

Isolated proximal tubular defect
Drugs (acetazolamide)
Genetic
Generalized proximal tubular defect (Fanconi's Syndrome)
Genetic
Hyperparathyroidism
Malabsorption syndromes
Drugs or toxins (ie, tetracycline or lead)
Associated with renal injury
Medullary cystic disease
Multiple myeloma
Renal transplant
Nephrotic syndrome

Bicarbonate administration is used to measure proximal bicarbonate excretion. If the serum bicarbonate is normalized by either intravenous or oral sodium bicarbonate administration and the fractional excretion of bicarbonate is greater than 15%, the patient has renal bicarbonate wasting. The fractional excretion of bicarbonate ($F_EHCO_3^-$) is calculated as

$$F_EHCO_3^- = \frac{\text{urine bicarbonate}}{\text{urine creatinine}} \times \frac{\text{plasma creatinine}}{\text{plasma bicarbonate}}$$

Renal bicarbonate wasting may be either drug-induced (carbonic anhydrase inhibitor) or the result of proximal renal tubule acidosis (RTA). There are many causes of proximal RTA. The interested reader should consult a standard nephrology text.*

After renal bicarbonate loss has been excluded, I separate patients into two groups based on the urine pH and the serum potassium (Fig. 1). If the serum potassium is less than 5 mEq/L in the presence of metabolic acidosis and a urine pH greater than 5.5, the patient has a defect in renal hydrogen ion secretion. This defect can be caused by a number of specific entities, but the general process is called distal renal tubular acidosis (Table 4). The reader interested in more information should consult a standard nephrology text.

In patients with fractional bicarbonate excretion less than 15% and elevated serum potassium during metabolic acidosis, it is important to look for a gen-

*See suggested reading list at the end of the text.

Table 4. Distal Renal Tubular Acidosis

Primary
Autosomal dominant inheritance
Secondary
Autoimmune diseases (Sjögren's, systemic lupus erythematosus)
Genetic disorders (Sickle cell anemia)
Disorders associated with nephrocalcinosis
Idiopathic hypercalciuria
Primary hyperparathyroidism
Hyperthyroidism
Medullary sponge kidney
Toxin-induced nephropathy
Amphotericin B
Lithium

eralized decrease in renal function, such as occurs with renal insufficiency or in systemic diseases with renal manifestation (Fig. 1).

If the BUN and creatinine are normal and no evidence for a systemic disease can be found, but the urine pH is greater than 5.5 in the presence of hyperkalemia and systemic metabolic acidosis, hyperkalemic distal renal tubular acidosis should be considered. Hyperkalemic distal renal tubular acidosis is most likely secondary to obstructive uropathy or a medication (ie, cyclosporine, amphotericin).

If the urine pH is appropriately acid (pH < 5.5), an abnormality in mineralocorticoid secretion or renal response should be considered. These can be evaluated by measuring serum cortisol and aldosterone.

As mentioned at the beginning of this paper, metabolic acidosis is frequently present in severely ill patients. Conditions producing metabolic acidosis are sometimes difficult to evaluate. I have developed this strategy during the last 25 years and continue to find it useful. I have organized it on paper in an algorithmic form in hopes that others may find the approach helpful.

ACKNOWLEDGMENTS

I thank Elizabeth S Patrick for her thoughtful editorial assistance and for carefully typing the final manuscript.

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Respiratory Care Practitioner: Carpe Diem!

Robert M Kacmarek PhD RRT

To me, the opportunity to deliver this lecture is a distinct honor and a turning point in the professional development of respiratory care. Too infrequently have we recognized individual respiratory therapists for their capabilities. Until now, Program Committee and Egan lectures have been provided by physicians. I don't want to minimize the contribution of physicians to respiratory care or their present active role and involvement in our profession because without their support we would not be here today, and I truly hope that our relationship with them continues to develop. However, I do believe it is important for us to look at each other and realize the capabilities of the person sitting next to us. Although I was chosen for this honor, numbers of people sitting in this room are qualified equally to be standing here instead of me. If it had been my choice, I would have chosen others to make this presentation. Three people who I feel deserve that recognition are Dean Hess, Rob Chatburn, and Rich Branson. All three have distinguished themselves in the field of respiratory care and equally deserve the recognition that has come to me.

Well, let me begin. I have been asked to discuss the future of respiratory care from the perspective of what we as practitioners need to do today. When a possible title for this presentation "Respiratory Care Practitioner: Carpe Diem!" was suggested, I must admit I had to look it up. I could not find the quotation "Carpe diem" in the medical library; I actually asked my wife to go to the local library to research the term. It took trips to a couple of different libraries to find the original quotation in an Ode by Horace in 23 BC. "Carpe diem" can be translated to "seize the day, trust the future as little as you may," or more literally "the future is purchased by the present."

Dr Kacmarek is Director, Respiratory Care Services, Massachusetts General Hospital and Assistant Professor, Department of Anesthesiology, Harvard Medical School—Boston Massachettes.

A version of this paper was presented by Dr Kacmarek as the Program Committee Special Lecture during the AARC Annual Meeting in Atlanta, Georgia, in December 1991.

Reprints: Robert M Kacmarek PhD RRT, Respiratory Care Services, Ellison 4, Massachusetts General Hospital, Boston MA 02114.

The Future

I want to focus this presentation on what we need to do today to define the future of respiratory care. Of course, no one can stand up here and predict the future. However, I have been coming to these meetings for a long time and every year as I go through the exhibit area, and every year as I listen to the different presentations, I am amazed at the continuing exponential growth in respiratory care. The difference between today and just 10 short years ago is incredible. The technology available, the topics presented by lecturers, the posters and the abstracts presented by you, the respiratory care practitioners, indicate to me that the future of respiratory care is bright and strong and enduring—with the role of the respiratory care practitioner continually evolving. If I were to look to the year 2000 and predict what would be going on in our intensive care units, I have to believe that the type of mechanical ventilators we are going to see next to a patient's bed are going to far exceed the capabilities of those currently available. All of the data that are available at the intensive care bed will be integrated via some type of computerized data acquisition system. In the past, a lack of data and lack of confidence in available data hindered the development of such a system. Indwelling, continuous arterial blood gas analysis is right around the corner. If this technology is half as good as suggested, the way we practice medicine in the ICU is going to change dramatically. As a consequence,

you and I will also have to change dramatically to keep up with the expectations of the medical profession. Extracorporeal gas exchange will come into its own in the next few years. I believe that extracorporeal gas exchange is going to be commonplace in many intensive care units, particularly for the very critically ill patient.

The respiratory care practitioner of tomorrow is going to have to be a skilled intensive care specialist. We must be able to manage patients clinically, but we must also have better computer skills and better engineering skills. We must be able to interact closely with all of the technology at the bedside.

Yes, I do believe we are going to see the loop closed—that is, closed-loop ventilation. We will be involved in programming ventilator systems, and, based on our programming of those systems, we will determine how the ventilator is going to respond to the patient data available from the myriad of monitors.

We will see dramatic growth in home care and rehabilitation—facets of medicine growing more rapidly today than most others. Our roles in rehabilitative medicine and gerontology need to be solidified and expanded.

Historical Development of Respiratory Care

To determine what we need to do to prepare ourselves for the future, we must review how we arrived at where we are today. It is important to see how rapidly we have grown as a profession and where we are today to appreciate the exponential growth that will continue in the future. As you are well aware, the Inhalational Therapy Association was founded in 1956 with 59 members; whereas Pat Dunne, in his 1991 President's report, stated that AARC membership is more than 31,000. Manpower studies indicate that more than 100,000 persons now practice respiratory care.

I have been practicing respiratory care for more than 25 years and during those years I have been called many things. The ones that I can mention are listed in Table 1. I began as an oxygen therapist and have gone through a number of title changes, as have many of you who have practiced during this period. These changes in title clearly reflect the level of responsibility and the level of growth of the profession. When we look back over the last 5 to 10 years at the growth in the respiratory therapy profession, we gain an appreciation of where we are and where we need to go if such growth is to continue into the next decade.

Table 1. Title Changes in Respiratory Care—1955-1991

Oxygen Orderly
Inhalational Therapist
Inhalation Therapist
Respiratory Therapy Technician
Respiratory Therapist
Respiratory Care Practitioner

Reviewing the history of respiratory care educational programs and respiratory care credentialing allows us to focus on our growth. The Board of Schools was established in 1956, with a total of 6 schools. Today, more than 450 respiratory therapy programs are in operation. Of these, 60-65% are Registry-level programs. In 1961, a short 30 years ago, there were only 12 Registered Therapists. I was registered in 1973, and my number is 1892. In 1968, the Technician Certification Board was established. The magnitude of this profession's growth is reflected by today's numbers (Table 2)—40,000 Registered Therapists plus 62,000 with the CRTT credential. There are 5,000 Certified Pulmonary Function Technologists and 1,800 Registered Pulmonary Function Technologists. Some 900 practitioners successfully completed the first specialty examination to be recognized as Perinatal-Pediatric Respiratory Care Practitioners. This proliferation of credentials and of credentialed personnel is testimony to the fact that we are part of an expanding, rapidly growing profession that is requiring greater and greater capabilities of each of its practitioners.

Table 2. Numbers of Credentialed Personnel in Respiratory Care

CRTT only	62,734
RRT and CRTT	40,865
CPFT only	4,893
RPFT and CPFT	1,848
Perinatal/Pediatric RCP	901
Total number of credentials awarded	111,241

We have developed in many, many areas. Dr William Burgin's comment when he received the Albert H Andrews Jr Memorial Award puts it all into perspective.

> Over the years the biggest factor that attests to the growth in respiratory care is that back in the 50s and 60s it was difficult to find a respiratory care department that was not in the basement of the institution. However, today it is rare that you will find a respiratory care department that is located in the basement.

Many of our departments are located right next to our intensive care units. Our own new department location is right in the middle of our three surgical units on the same floor. In the various institutions that I've visited, a similar scenario exists. We are considered essential to the management of patients, particularly those patients who are critically ill.

Problems Affecting Future Growth

It is important for us to discuss some of the problems and concerns that must be addressed if we are to continue to grow at the same pace. As a profession, we have an identity crisis. Problems long ignored in education and credentialing must be corrected. Attrition is hurting our continued growth. Some of our best therapists are leaving respiratory care—becoming managers or going into other professions. In addition, we continue to have manpower shortages.

Our identity crisis is the single factor that hurts us most as a profession. How many times have you been asked, "What is a respiratory care practitioner?" or "What is a respiratory therapist?" Everybody knows (or thinks he or she knows) what a nurse does, everybody knows what a laboratory technician does, but people still don't know—even people in the hospitals in which we work—don't understand what a respiratory therapist does. Variation in role definition from hospital to hospital adds to the identity crisis. In any given city, the therapists in one institution play true consulting roles in the management of patients—involved in decision making, participating in rounds at the bedside, a part of the patient-care process. In the institution across the street, the therapists maintain equipment, set up ventilators, move them into the ICU, plug them in, and make sure they are operational, but somebody else actually takes care of that patient-ventilator system on a moment-to-moment basis. Therapists may come in and check the ventilator, but they don't play the role of consultant, they don't play the role of a true therapist. They are relegated to functioning purely as equipment technicians. Unfortunately, this scenario exists nationally, regionally, and locally. This gross variation in role definition is a critical variable that is adversely affecting the growth of respiratory care.

Today, all new entering practitioners must graduate from a respiratory therapy program. However, institutional differences and the motivation (or lack thereof) created within institutions determines the growth and the development of individual practitioners. In how many institutions is there no incentive for the individual to become registered after graduation? In how many is there no incentive to take advantage of educational opportunities that enhance performance? In order to continue to grow and develop as a profession, we must eliminate complacency. Status quo doesn't work! One either moves ahead or falls backward. And if we are not aggressively moving forward, we as a profession will surely move backward. Each of us must continually encourage new graduates to become Registered Therapists.

When therapists call us about a job, we ask them their background. When they say that they graduated from a Registry school 5 years ago but are not registered and we respond that we only hire new graduates or Registered Therapists, they are amazed. If you have not completed your education by becoming a Registered Therapist, you have not demonstrated your commitment to the profession of respiratory care. We all share responsibility for creating the incentive and providing the motivation that ensures that all graduates become registered.

It is time for us to take a critical look at our two-level system of education. When we introduced the technician level, it was essential for meeting the manpower needs of a rapidly growing profession. Today's growth is different from that 20 years ago. We are not as much in need of numbers, although manpower shortages do exist, but we do require large numbers of highly trained therapists. I believe that the present bi-level educational system is not

meeting our professional growth requirements. One can go into institutions in which therapists and technicians work next to each other performing the same job but usually receive different salaries. I believe we are doing an important disservice to students by directing them to Technician Certification programs, rather than to Registry programs that prepare them for what I—and for what I believe most managers—expect from a school graduate. Considering the scope of respiratory care, the technology, and the respiratory care procedures we are expected to perform, how can we adequately educate students in a 12-month technician program? The demands that the medical profession is placing on us and the demands that every one of us should have of the other requires a change in this system.

In addition to problems regarding a bi-level educational system, the recent changes incorporated by the Joint Review Committee for Respiratory Therapy Education (JRCRTE) have created enormous problems within educational programs, redirecting the focus from actual student education. The change in approach from process to outcome is clearly appropriate, but in making the change we have failed to provide educators with the information they need to allow for appropriate development of their programs. Some middle ground must be established between extremes. It is my understanding that this need is now being met. But, for the last 18 months to 2 years, it has been difficult for educational programs to focus on their primary reason for being—education of respiratory care practitioners—rather than focusing on maintaining accreditation of their programs.

Professional attrition and manpower shortages are problems for every one of us. We need mechanisms in respiratory care designed to better establish career ladders that keep qualified practitioners in the profession. As I talk to people, I find it distressing that very few will tell me that they want their children to become respiratory care practitioners. Many have no pride in the fact that they are respiratory care practitioners. We need to have more pride in what we do, and we need to keep the best respiratory care practitioners in the field by making the profession more exciting and more academically demanding. We have to position ourselves in our individual institutions to be ready to assume roles that would allow us to be part of new and advancing technology. Extracorporeal membrane oxygenation (ECMO) is an example of where we are failing to do this. Table 3 presents the data released this fall from the International ECMO Registry. You can see that large numbers of patients have been managed with ECMO. You will also notice that many centers provide ECMO. However, the number of those centers at which this method of maintaining gas exchange is technically managed exclusively by respiratory care practitioners totals only four. In some programs, practitioners from a number of professions are involved, but in a large number of programs respiratory care practitioners are not involved at all. This is unacceptable. We have to aggressively look at involvement in these types of innovative approaches to maintaining overall gas exchange if we are to maintain our best practitioners and develop professionally.

Table 3. ECMO International Summary, Fall 1991

Patients	Cases	Survived	Total Centers*
Neonatal	5,479	83%	79
Pediatric	285	47%	53
Cardiac (P+N)†	494	46%	62
Adults	44	43%	7

*4 centers are manned solely by respiratory therapists.
†P + N = pediatrics & neonatal.

As a profession, as individuals, we are at a critical point. We need to look at ourselves carefully and look at what we need to do to position ourselves for the future. There is no question in my mind that we are capable of assuming the role that is developing, but we cannot be complacent and expect it just to come to us. Every one of us wants professional respect. Every one of us wants some type of expanded career opportunity. But, each and every one of us must be willing to put in the time and effort needed to further develop and be willing to support our profession and to assist it in initiating the changes that are necessary to enter the next century in a position to maintain the impact that we have today. We need to aggressively market respiratory care. We need to encourage credentialing at the highest possible level by all of our practitioners. For those who are presently technicians, we need to provide incentives for them to become therapists. The goal for every one of us should be

to assist other practitioners in attaining the higher levels of distinction within the profession.

As you are well aware, in each of our institutions respiratory care is judged by its least common denominator. Those practitioners who are least capable project to the rest of the medical staff and to other health care workers the capabilities of the respiratory care profession in general. Members of other professions don't look at the best therapist; they don't look at the average; they focus on that least common denominator. We must increase the least common denominator.

Changes Needed Today

We need to be more involved in developing the scientific basis for respiratory care. I'm quite pleased at the growth that has occurred in the last 4-5 years. The OPEN FORUM presentations at the AARC's 1991 Annual Meeting attest to our scientific growth. We are performing high quality respiratory care research. We are actively involved with physicians in providing a sound basis for respiratory care. This activity needs to develop to an even greater level, involving greater numbers of respiratory care practitioners.

We need to plan for an expanded educational base. As I indicated earlier, it is time for a single-entry educational system. We must establish single-entry at the Registered Therapist level. I propose that we phase out all Technician-Certification programs over a 5-year period. As a profession, we need to be moving forward and creating educational systems that prepare all of our practitioners to achieve the level that will be expected of us in the years to come. In addition, we need to expand baccalaureate-level education.

Therapists ask, Why aren't we paid the same as physical therapists, as occupational therapists, as registered nurses? One of the major counters to equal pay that I hear from administrators and personnel departments is, "Look at the educational preparation expected of the physical therapist, of the occupational therapist, of the speech therapist." The entry level for physical, occupational, and speech therapists is a baccalaureate or a master's degree; even registered nurses have a minimum of an associate's degree, whereas our entry level is a 1-year non-degree program. How can we expect hospital administrators to view us as equivalent when educational requirements for entry-level respiratory care are compared to those of other groups. I'm not saying that the length of training is the only factor to consider, but it makes it easy for administration to counter all arguments regarding salary by simply comparing educational preparation. I do believe that we are as capable as any of the other professions, but our educational requirement makes it easy for people to say "No, you are not equivalent." In addition, we need to expand what we are teaching. As I indicated earlier, I think we as therapists need to have a better basic understanding of engineering techniques, computer technology, extracorporeal gas exchange, and cardiopulmonary diagnostic techniques.

Manpower has to be viewed as a problem by every one of us if we are to continue to develop as a profession. Managers and educators need to work together to keep students in school by assisting underprivileged youth to complete educational programs. We, in Massachusetts, for whatever reason, are fortunate in that we don't have a manpower shortage, but I'm told that's not true for the rest of the country. In the process of eliminating manpower shortages, we set up programs to provide scholarships that entail an employment commitment once school is completed. We need to creatively develop similar mechanisms to assist students with school and to allow educational access for those considering respiratory care. In our institution, we bring high school students into our departments. We have not been as aggressive in visiting schools as we could be, but we have succeeded in bringing students into the hospital setting and introducing them to respiratory care. We can all market respiratory care by similar activities.

Solving retention in the field of respiratory care is not easy. Every one of us has to go back and look at what is wrong in his or her own department. My department has problems. I need to address some of the problems that affect retention, that create a poor attitude in some of my staff. Each of our departments can be improved. There is something wrong with the way many departments are run. We need to look critically at what it is we have to change within our own environments to enhance the working conditions for our employees so that

we can retain staff. I find respiratory care exciting! I must in some way make my staff find respiratory care exciting, and I believe that every manager of every department should have a similar goal. If your staff doesn't enjoy working with you, the likelihood is that they are not going to work very long. If we are not creating a milieu in which staff can grow and develop and feel as if they are practicing as therapists, they will move on to a different department or a different profession.

We need to aggressively pursue performance of whatever new therapies, whatever new technologies enhance the practice of respiratory care. Many departments are embracing new technology; however, as illustrated by the ECMO data, many are not. Manpower shortages are preventing departments from getting involved with emerging programs. Mine is one of those institutions in which ECMO is done by respiratory care practitioners. We began the ECMO program without a single dedicated staff position. We still have not received additional staff to perform ECMO. My staff has been willing to participate in the ECMO program, willing to go on call, willing to work overtime shifts, in order to allow Respiratory Care to be the exclusive group providing ECMO. I am proud of the effort that they have made over the years in the ECMO program. This type of commitment can be found in most groups if you provide the opportunity to grow and develop.

There is no reason why we cannot embrace new therapies. However, we may have to give up old therapies that do not require Registered Therapists. For example, how necessary is it for a Registered Therapist to check a nasal cannula twice a shift? Or, do I need to have a Registered Therapist do ventilator rounds hourly on stable patients? We may be creating manpower issues by our lack of creativity and our unwillingness to change behavior that has continued for years. Managers must critically evaluate their departments and modify approaches to be most cost-effective if we are to continue to develop. Work load can be cut to allow us to become involved in new therapies, but we must be willing to look critically at what we are doing and be willing to change the way we perform respiratory care, in order to progress.

The opportunities for the future of respiratory care are limitless. I'm convinced after 25 years as a respiratory therapist—progressing from pushing oxygen cylinders on a daily basis to my present position—that we as a profession can develop to whatever level we choose. If we address the problems outlined, I believe we are capable of grasping that opportunity and meeting the challenges, but it requires the effort of every single respiratory care practitioner.

I leave a few thoughts for individual therapists. You need to invest in respiratory care. You need to become true professionals—not factory workers working in hospitals. We all need to be part of the solution—not part of the problems that exist today.

And, the last thing is a quote from one of the people who received an award at this 1991 AARC Annual Meeting. Connie Jones, a Registered Therapist who received the Outstanding Affiliate Contributor Award, said, "It's the little things that make the difference." If each of us makes a small investment on a daily basis for the betterment of the respiratory care profession, we will all be able to "seize the day."

"Carpe diem."

Contributions to this section welcome. send double-spaced typed material (and illustrations if permitted) to Historical Notes, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593. Identify source of material and include your name, credentials, and address.

Listening to the Chest in the 1930s*

Auscultation of the Lungs

In listening to the lungs, we have two methods at our disposal, immediate auscultation which consists in placing the ear directly against the chest wall, and mediate auscultation which consists in listening through a stethoscope. While the mediate method is the medthod universally employed, the student should become familiar with the immediate method as well. The occasion may arise when the physician through unforeseen circumstances does not have his stethoscope with him. In examining babies or children the appearance of the stethoscope may excite the fear of the patient who does not object to the examiner placing his ear against the chest. Again, just as the examiner always has his fingers with him for percussion, so he always has his ears for auscultation.

Stethoscopes

The earliest type of stethoscope was the monaural stethoscope devised by Laennec. This stethoscope, usually made of wood, is 1 foot in length, has a funnel-shaped enlargement on the end which is placed on the chest, and a flat flange on the end which is applied to the ear (Fig. 200†). This type of stethoscope, while somewhat of a curiosity in this country, is still extensively employed in European clinics, where physicians still champion its superiority and point to its use for more than a century (Fig. 201). There is no question of its value in the hands of those familiar with it. Most, if not all, of the great discoveries in auscultation have been made with this instrument. Yet it is certainly less convenient to use than the binaural type. Thoroughly to explore various regions of the chest with this stethoscope sometimes requires an almost acrobatic agility on the part of the examiner. The American physician usually complains that with the monaural stethoscope he hears too little, to which his Continental colleague retorts that with a binaural stethoscope he hears too much. . . .

Hippocratic Succussion

This classical sign was first described by Hippocrates* in empyema. "Set the patient on a stool, which is not wobbly, someone should hold his hands, then shake him by the shoulders and listen to see on which side a noise is heard." (Fig. 217).

Laennec was the first to recognize that Hippocratic succussion is due to the presence of both air and fluid in the chest and to point out, that even a large amount of fluid in the chest produces no splashing sound, when the patient is shaken, unless there is also air present. Air is necessary to produce a splashing sound. A bottle full of water can be shaken violently without

* These excerpts are from the book Physical Diagnosis, 3rd edition, by Ralph H Major MD, Professor of Medicine at The University of Kansas. The publisher was WB Saunders Company of Philadelphia. The book first appeared in 1937; these excerpts were from the edition printed in 1948. Larry Boller PhD RRT, Director of Allied Services at The Monroe Clinic, Monroe, Wisconsin, discovered this material and made it available to RESPIRATORY CARE. Reprinted with the permission of WB Saunders Company.

†Editors note: No figures were available for reprinting.

the production of a splashing sound, a bottle half-filled with water gives a splashing sound when agitated violently.

This splashing sound is often heard by the patient when he walks or climbs stairs. At other times he feels the displacement of the fluid caused by change in posture. Morgagni described a patient who "said that when she moved her chest she felt a splashing within."

The succussion is best elicited by following the ancient method of Hippocrates,‡ shaking the patient by the shoulders and listening with the ear or stethoscope against the chest. The splashing sound caused by disturbing the level of fluid is easily heard.

‡Editor's note: Hippocrates lived around 600 BC.

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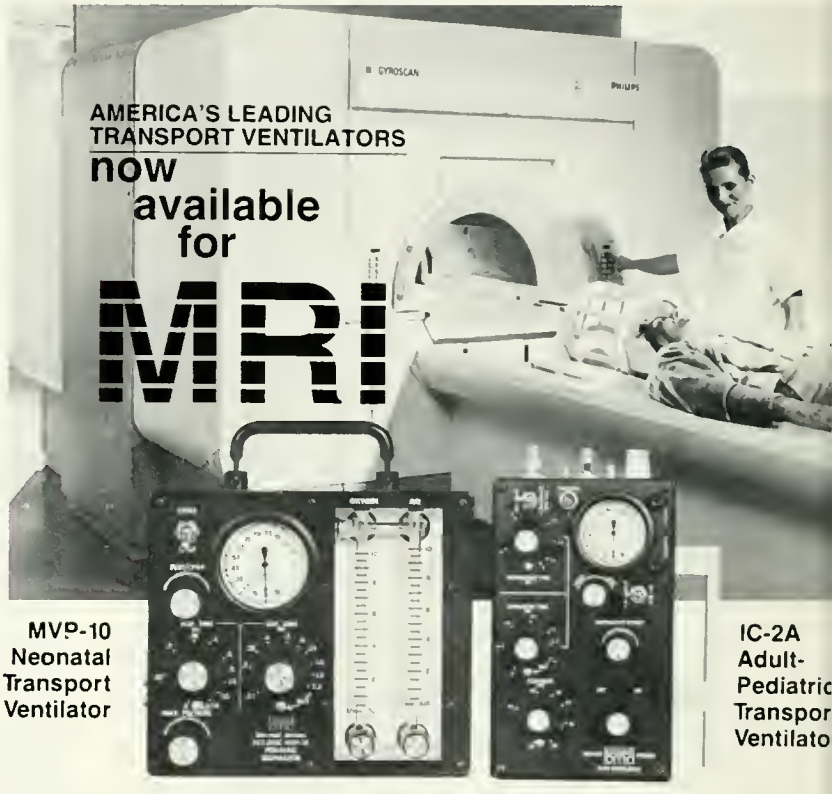

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Foul-Smelling Sputum, Malaise, and Night Sweats

Digpal Chauhan MD

A 60-year-old man was admitted with the chief complaints of purulent and foul-smelling sputum, hemoptysis, malaise, weight loss, and a 2-week history of fever and night sweats, with some right-side pleuritic chest pain.

The patient denied exposure to pulmonary tuberculosis. He had been a pack-a-day smoker for many years but gave no history of ethanol abuse. His oral temperature was 99.2°F. Examination of the mouth suggested poor oral hygiene and neglect and revealed severe dental caries with periodontitis (Fig. 1). Percussion and auscultation of the thorax revealed dullness and bronchial breath sounds in the right upper lobe (RUL). Digital clubbing was present. The posteroanterior and lateral radiographs taken at that time are shown (Figs. 2A and 2B).

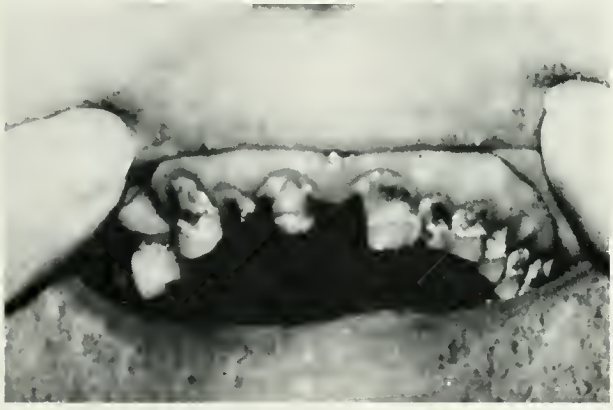

Fig. 1. Photograph of mouth of patient complaining of foul-smelling sputum, malaise, and night sweats.

Questions

Radiographic Findings: What abnormalities are seen in Figure 2?
Diagnosis: What is the most probable diagnosis?
Further Tests: What further testing is indicated?
Therapy: What therapy is indicated?

Answers and Discussion on Next Page

Dr Chauhan is Chief of Pulmonary Medicine, VA Medical Center, Leavenworth, Kansas.

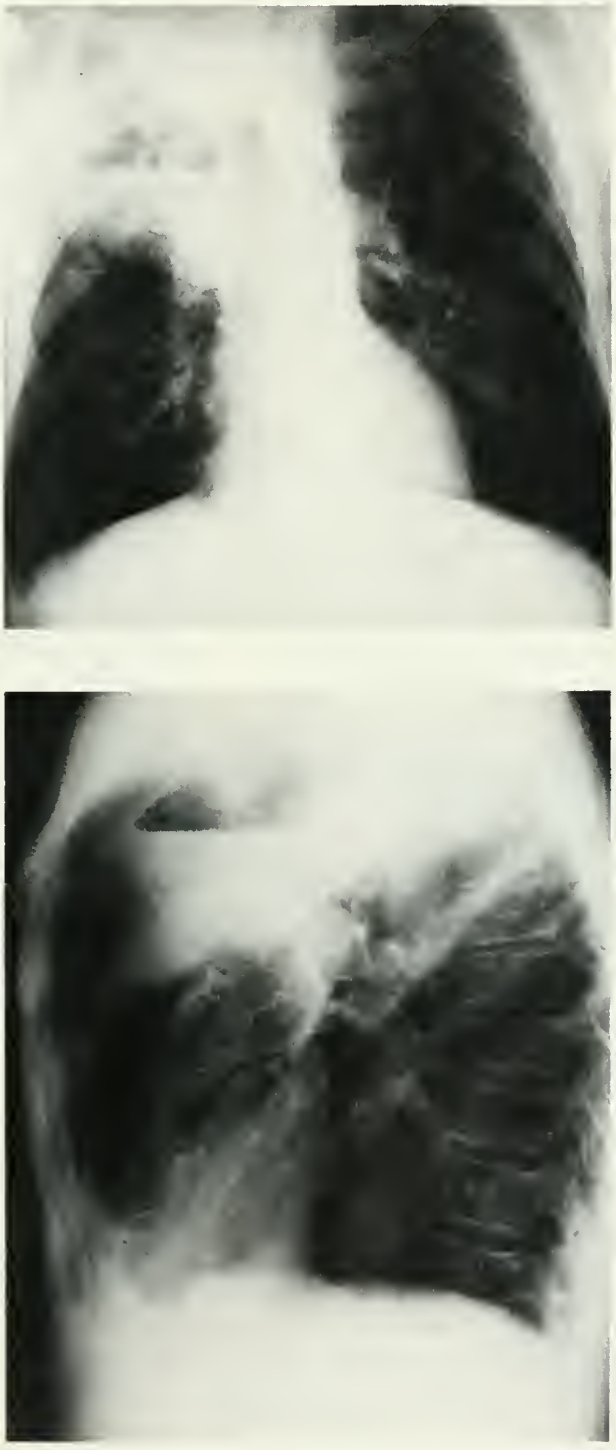

Fig. 2A. Posteroanterior radiograph of patient on admission. 2B. Lateral radiograph of patient on admission.

Answers

Radiographic Findings: An air-fluid level is seen in the anterior segment of the RUL with infiltrates suggesting additional disease in the apical and posterior segments as well.

Probable Diagnosis: The air-fluid level in the posterior segment is indicative of lung abscess resulting from aspiration.[1]

Further Tests: The infiltrates in the anterior and apical segments cannot be explained by aspiration alone. The sputa sent for acid-fast bacillus (AFB) smear and cultures revealed *Mycobacterium tuberculosis*. Sputum cultures also grew *Enterococcus*. Bronchoscopy ruled out any bronchial obstruction in the anterior segment or other endobronchial lesion.

Therapy: Aggressive antibiotic therapy is indicated for the mixed infection giving rise to the lung abscess and for the *M. tuberculosis* infection. The patient was treated for 2 weeks with gentamycin, for 8 weeks with clindamycin, and for 9 months with antituberculosis drugs. The chest film showing resolution of the abscess cavity at hospital discharge is seen in Figure 3.

Fig. 3. Posteroanterior radiograph of patient on release from hospital after treatment.

Discussion

Lung abscess is a suppurative infection that destroys lung parenchyma to produce one or more large cavities with air-fluid levels. A similar process with many small cavitations (< 2 cm in diameter) has been designated as necrotizing pneumonia. The distinction between the two is arbitrary because lung abscess and necrotizing pneumonia are different expressions of the same fundamental pathologic process. Aspiration is the predisposing factor for lung abscess and may be related to an altered level of consciousness as in alchoholism, drug overdose, and seizure disorder.[2] The organisms producing necrotizing pneumonia are similarly aspirated from the pharynx, and the difference in presentation may well be in part due to the size and dispersal of the inoculum.

A study by Huxley et al[2] using sensitive radioactive tracer techniques demonstrated that about 45% of healthy people aspirate and about 70% of patients with depressed sensorium aspirate during sleep.

Periodontal disease, or gingivitis, as seen in our patient predisposes to lung abscess or necrotizing pneumonia. Lung abscess is rare in edentulous persons, and lung abscess in an edentulous person should lead to a diligent search for bronchogenic carcinoma.[3]

Oropharyngeal secretions in adults contain a high concentration of bacteria—as high as 10^9 organisms/mL in saliva. The ratio of anaerobes to aerobes is approximately 5:1. Material from the gingival crevices in patients with periodontal disease contain 10^{11} to 10^{12} bacteria/mL,[3,4] with an anaerobe-to-aerobe ratio of 1,000:1.

The bacteriology of anaerobic pleuropulmonary infections is complex and, unlike infections caused by pathogenic aerobes, often includes many different organisms. Cultures of transtracheal aspirates in cases of lung abscess and anaerobic necrotizing pneumonia yield, on the average, 2.4 different anaerobes.[2,3] The most common pathogens include the gram-negative bacilli, *Bacteroides melaninogenicus*, and *Fusobacterium nucleatum*, and gram-positive *Peptococcus* and *Peptostreptococcus*. In one third of the cases, aerobes are also recovered, but their pathogenic significance in this setting is unknown. In a prospective study of 26

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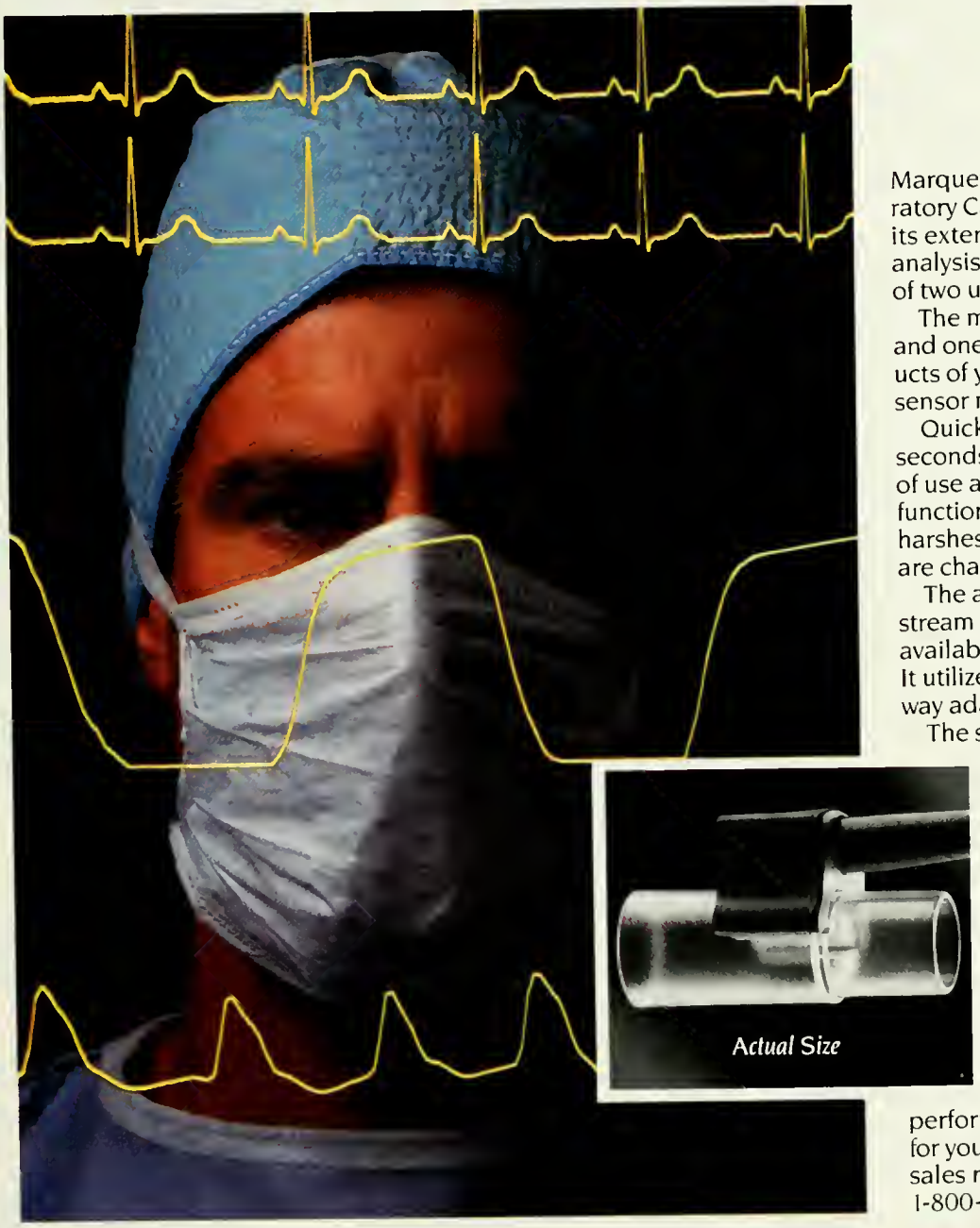

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patients with lung abscesses, Bartlett et al[1,4,5] recovered anaerobic bacteria in 24 of the 26 tracheal aspirates.

Anaerobes may cause four types of chest infection: the solitary lung abscess, necrotizing pneumonia, nonnecrotizing pneumonia, and empyema.

A variant of necrotizing pneumonia is a massive pulmonary gangrene. In this rare condition, a large area of lung, usually an entire lobe, becomes necrotic and is sloughed—giving the appearance on radiograph of a large chunk of solid tissue within a large cavity (a floating iceberg).[3] Pulmonary gangrene, unlike more common anaerobic infections, is due to septic endarteritis involving arteries supplying the affected lobe. Although anaerobes are most likely involved, mixed aerobic-anaerobic infections may be important in some cases, and in some cases aerobic bacteria alone may be responsible.

Patient symptomatology varies from productive cough with putrid, foul-smelling sputum to hemoptysis to malaise and fever. Patients can also experience weight loss and anemia. Patients with anaerobic necrotizing pneumonia can be quite ill and have a temperature up to 103°F. The white count usually shows a significant shift to the left, and one third of the cases also have empyema.[3]

Anaerobic pleuropulmonary infections are suspected on clinical grounds. Bacteriologic proof of anaerobic lung infection requires a specimen for culture obtained by transtracheal or transthoracic-needle aspiration that bypasses the contaminated oropharynx, but these techniques are rarely necessary for management of anaerobic pleuropulmonary infections. In the presence of pleural effusion, fluid can be tapped easily and pleural fluid be sent for gram stain, cultures, and pH and glucose determinations. Recent experiences with cultures from specimens obtained via protected brush bronchoscopy have been encouraging.[3,6]

Therapy with clindamycin or metronidazole supplemented with penicillin-G is the treatment of choice for serious anaerobic pulmonary infections.[3,4] Metronidazole has superb activity against all anaerobes (but not against aerobes or microaerophilic streptococci) and has not been proven effective as a single agent for lung abscess.[3,7] Increasing numbers of cases are becoming resistant to penicillin, primarily due to the presence of beta-lactamase-producing *Bacteroides*. Penicillin is still favored as initial therapy for patients who are mildly to moderately ill.[8]

External percutaneous drainage may be required when a patient with a large lung abscess remains ill despite appropriate antibiotic therapy. Adequate drainage is the cornerstone of management of empyema, although antibiotics should also be given. Percutaneous tube thoracostomy is effective in establishing drainage and thereby decreasing toxicity in the majority of patients.[3,9]

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INSTRUCTIONS FOR AUTHORS

In addition to case reports strictly involving pulmonary medicine radiography, case reports involving adult, pediatric, and neonatal critical-care radiography can also be submitted to the "Test Your Radiologic Skill" corner. However, all case reports should relate somehow to respiratory care. Illustrative radiographs may be of routine chest exams or of other less-common exams such as digital subtraction or computerized axial tomography.

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AARC & AFFILIATES

March 18-19 in Newport, Rhode Island. The Rhode Island Society for Respiratory Care presents The Rhode Island Respiratory Care Symposium, "The Newport Challenge—The Tradition Continues." Contact Skip Bangley RRT, PO Box 9051, Providence RI 02940-9051. (401) 456-4174.

April 8-10 in Chattanooga, Tennessee. The TSRC presents its Annual Convention and Exhibition at the Chattanooga Choo-Choo. The program includes day-long workshops on Test Taking Techniques for students and "Polishing Your Professional Image" for practitioners and featured speakers Cheryl Brown; Bob Demers RRT; Myrna Dolovich PhD; Ross Duteau RRT; David Geller MD; John Hiser MEd RRT; Jane Isbell; Damon Lawson RRT; John Marini MD; Neil MacIntyre MD; Tommy Rust MEd RRT; and Novieta Wilson. Social events include the annual Sputum Bowl competition and theme party. Contact Colleen Schabacker (615) 384-1569.

April 13-15 in Philadelphia, Pennsylvania. The PSRC presents its 27th Annual Conference and Exhibition, "Foundations and Discoveries," at the Adam's Mark Hotel. Contact Betsy Schneck (215) 829-3578 or Kathy Yandle (215) 453-4517.

April 15-17 in Des Moines, Iowa. The Iowa Society for Respiratory Care presents the 14th Annual Iowa Lung Conference at the Des Moines Marriott Hotel. Topics include interpersonal relations in the work place, preparation for the CRTT and RRT exams, CLIA update, job interviewing, non-traditional bronchodilator delivery, mechanical ventilation, and nutritional support. Special speaker is Bob Gibson, baseball hall-of-famer. Contact Mike Wheeler RRT, Respiratory Care, Iowa Lutheran Hospital, Des Moines IA 50316. (515) 263-5108.

April 16-17 in Lawrence, Kansas. The KSRC presents its 15th annual education seminar. Topics include pulmonary rehab, professionalism, the resurgence of TB, and asthma. A golf tournament and Sputum Bowl are scheduled for the 15th. Contact Frank Hart, Pulmonary Services, Shawnee Mission Medical Center, 9100 West 74th St, Shawnee Mission KS 66201. (913) 676-2175.

April 21-24 in Cincinnati, Ohio. Ohio, Kentucky, and Indiana present the 19th Annual Region II for Respiratory Care meeting at the Albert B Sabin Convention Center. The event begins April 21 with four concurrent specialty programs: alternate site ventilation, people skills, lab quality assurance, and laughter in nicotine addiction. The regular meeting then continues April 22-24. Other events include over 100 vendor exhibits and Sputum Bowl competitions. Contact Michael Moore (513) 221-3050.

April 29-30 in Waterbury, Connecticut. The CSRC presents the 11th Annual Super Symposium at the Sheraton Hotel. Topics include ultra high frequency jet ventilation, ECMO, pressure control ventilation, and rehab. Social events include Wallyball and a dance. A golf tournament is planned for the 28th. Contact Dan Madison, CSRC, PO Box 3256, Enfield CT 06083. (203) 524-3212.

April 29-May 1 in Osage Beach, Missouri. The MSRC presents its 21st Annual Educational Seminar at the Tan-Tar-A Marriott. Topics cover a wide range of subjects, including care of the caregiver, CQI, and respiratory protocols. Contact Wilma Alexander (816) 276-4222.

May 3-5 in Bellevue, Washington. The 19th Annual Pacific Northwest Regional Respiratory Care Conference is slated for at the Bellevue Red Lion Inn. Topics cover pediatric, management, and critical care issues. A special guest speaker addresses the establishment of a 4-year respiratory care program in Washington state. Call Richard Larson (206) 880-4585.

May 7-9 in Los Angeles, California. The CSRC Chapter IV presents the 2nd Neonatal/Pediatric Pulmonary Care Conference and Golf Tournament. Topics at this 2-day conference include PFTs, HFV, asthma, synchronous infant ventilation, PEP & autodrainage therapy, cardiac surgery update, and surfactant controversies. The cost is $45/day with lunch included. Contact Kelvin MacDonald, Kaiser Permanente, (213) 667-6635.

May 28-29 in Laurel, Maryland. The MD/DC Society for Respiratory Care holds its 2nd Annual "Conference by the Tee" at Patuxent Greens Country Club. Topics include PC/IRV, ACLS and the RCP, CLIA validation protocols, continuous-flow ventilation, and trends in mechanical ventilation. Contact Joe Lynott (202) 877-1064.

OTHER MEETINGS

March 23-25 in Ft Lauderdale, Florida. The Cleveland Clinic Foundation sponsors a continuing education program entitled "Pulmonary & Critical Care Medicine" at the Pier 66 Hotel. Contact Cleveland Clinic Foundation, Dept of Continuing Education, 9500 Euclid Ave, Cleveland OH 44195-5241.

March 26-28 in Destin, Florida. The Pediatric Pulmonary Centers at the University of Alabama Birmingham, Tulane School of Medicine, and the University of Florida sponsor "Practical Issues in Pediatric Home Care: An Interdisciplinary Approach" at the Sandestin Beach Hilton. Contact Charlene Rhoades (205) 939-9583.

April 6 in Cincinnati, Ohio. A 1-day course entitled "Understanding Lung Function Tests" is sponsored by the University of Cincinnati. Contact Occupational Pulmonary Services (513) 558-1234.

April 9-10 in San Diego, California. The California Society for Pulmonary Rehabilitation (CSPR) presents the 3rd annual state meeting, "Charting New Horizons in Pulmonary Rehabilitation" at the U.S. Grant Hotel in San Diego. Contact Mary Jo Zanella-Goldzimer, program chairperson, 3335 Country Rose Circle, Olivehain CA 92024. (619) 740-4584 (beeper).

May 16-23 on a Caribbean Cruise. Come aboard the new Costa Classica for a "Calm Seas Ahead" voyage to Jamaica, Grand Cayman, Cozumel, and Playa del Carmen/Cancun. For information, contact Dream Cruises (714) 636-6660 or 800-462-3628. Write 10882 La Dona Ave, Garden Grove CA 92640.

June 22 in Cincinnati, Ohio. A 1-day course entitled "Spirometry Refresher —Update" is sponsored by the University of Cincinnati. This course reviews recent changes in spirometry testing standards, guidelines, interpretation techniques. Contact Occupational Pulmonary Services (513) 558-1234.

Books, Films, Tapes, & Software

Problems in Respiratory Care: Pathophysiology and Treatment of Inhalation Injury, edited by William G Cioffi Jr MD (Guest Editor) and Richard D Branson AS RRT and Neil R MacIntyre MD (Editors-in-Chief). Hardcover, illustrated, 119 pages. Philadelphia: JB Lippincott Co, 1991. $60.00.

This text, designed as an overview of the pathology and treatment of severe smoke inhalation injury, is targeted for the respiratory therapist. In general, the various chapters are adequate, although somewhat superficial, reviews of the subject material—chapters dealing with etiology, diagnosis, pathophysiology, management, resuscitation anesthesia, and long-term sequelae of inhalation injury.

In general the book is poorly illustrated; each chapter contains, on the average, only two figures. A number of figures are of poor quality, some to the degree that they do not depict what the figure caption describes (Chapter 1-Fig. 1; Chapter 4-Fig. 3; and Chapter 8-Fig. 1). Also, the choice of figures and tables is questionable (eg, the chapter dealing with pathophysiology doesn't contain a single figure illustrating pathologic changes in the lung and the sole table in Chapter 3 is used to describe the toxic components of smoke).

In the chapter dealing with pathophysiology, the sections on asphyxiants, chemical damage, and (especially) specific irritants are well done; however, the section on thermal damage is misleading. Although it appears that it is the consensus of the medical community that the proximal airway is such an effective heat exchange system that the pulmonary parenchyma is almost never damaged by heat, the author cites a study (Reference 33) to support the possibility of heat-induced lung damage that, in fact, describes the difficulty in delivering a heat load sufficient to damage the lower lung. The section of this chapter characterizing smoke pathophysiology would be confusing to any reader not well versed in the discipline of pulmonary physiology. In addition, the reader is forced to absorb numerous complex concepts entirely from the text because, as stated previously, the single table describes the toxic compounds contained in the smoke. This section skims over such complex subjects as neutrophil and complement activation, eicosanoid biochemistry, the surfactant system, the physiology of lung-fluid balance, and the animal model of lung lymph cannulation that is utilized to assess pulmonary vascular endothelial permeability. In contrast, I found the chapters dealing with fluid resuscitation and infection very well done, with excellent figures found in the fluid-resuscitation chapter.

The book closes with chapters on anesthesia and long-term sequelae of smoke inhalation that are of little benefit to the respiratory therapist. The challenges of proper anesthesia in the inhalation patient may be of great interest to the anesthesiologist, but I believe are of little practical interest to the respiratory therapist. On Page 389, in the chapter characterizing long-term effects of smoke inhalation, the author states, "Chronic sequelae directly attributed to the smoke are rarely encountered," which makes one wonder why an entire chapter would be devoted to a problem that rarely occurs.

In summary, this text reviews several topics germane to smoke inhalation injury. The chapters are well referenced, although, for the most part, poorly illustrated. The material dealing with the pathophysiology of acute lung injury is not adequately explained and is therefore difficult to understand. The sections on fluid resuscitation and infections are well done, but the chapters on anesthesia and long-term sequelae of smoke inhalation have little practical value to the practicing respiratory therapist. Since this text does discuss a number of important topics and is well referenced, I would recommend it as a brief overview of the subject to be used as a base for more extensive study.

Gary F Nieman BS
Research Scientist
SUNY Health Science Center
Syracuse, New York

Shortness of Breath: A Guide to Better Living and Breathing, 4th ed, by Kenneth M Moser MD, Andrew L Ries MD, Dawn E Sassi-Dambron RN BSN, Birgitta K Ellis PT, Trina M Limberg BS RRT, and Roseann Myers RN. Paperback, 118 pages, illustrated. Chicago: Mosby-Year Book, 1991. $13.50.

The fourth edition of this book was written by a multidisciplinary team as a reference for the patient with chronic lung disease. While the book remains essentially unchanged in content, a new chapter on pulmonary rehabilitation has been added. This is an important addition to the book because it gives the reader knowledge of availability of additional medical treatment that is not covered by the scope of this book. The authors state that a motivated and committed person can receive measurable benefits from a pulmonary rehabilitation program (such as increased exercise capacity, improved

ability to perform activities of daily living, decreased sensation of shortness of breath, improved quality of life, and decreased time spent in the hospital).

The text continues to be very readable, and the illustrations and content have been updated to more state-of-the-art therapy and equipment usage, especially noticeable in Chapter 5, "Breathing Paraphenalia." Information about spacers and extenders has been added along with a more detailed description of oxygen delivery systems, transtracheal catheters, and oxygen conserving devices. IPPB as a means of delivering aerosol medication has been deleted.

The book ends on a positive note with what can be expected in the future for the treatment of chronic obstructive pulmonary disease (COPD). Chapter 19, "A Look to the Future," reports that as a result of continuing research, an alpha$_1$-proteinase inhibitor for the rare individual with emphysema due to alpha$_1$-antitrypsin disease has been developed, new medications have been introduced that relieve spasm of bronchial tubes or inflammation of the mucous membrane, and lung transplantation may be an alternative to be considered for some with this disease.

The book, one of the simplest of its type to understand, is a complete handbook for the COPD patient's use as an educational tool at home or in conjunction with a formal pulmonary rehabilitation program. Many of our patients at Baylor University Medical Center tell us that the book is very helpful and that they refer to it to remind themselves of what they should do to help them live with COPD.

Pamela Corpier Marsh BS RRT
Coordinator
Pulmonary Rehabilitation
Baylor University Medical Center
Dallas, Texas

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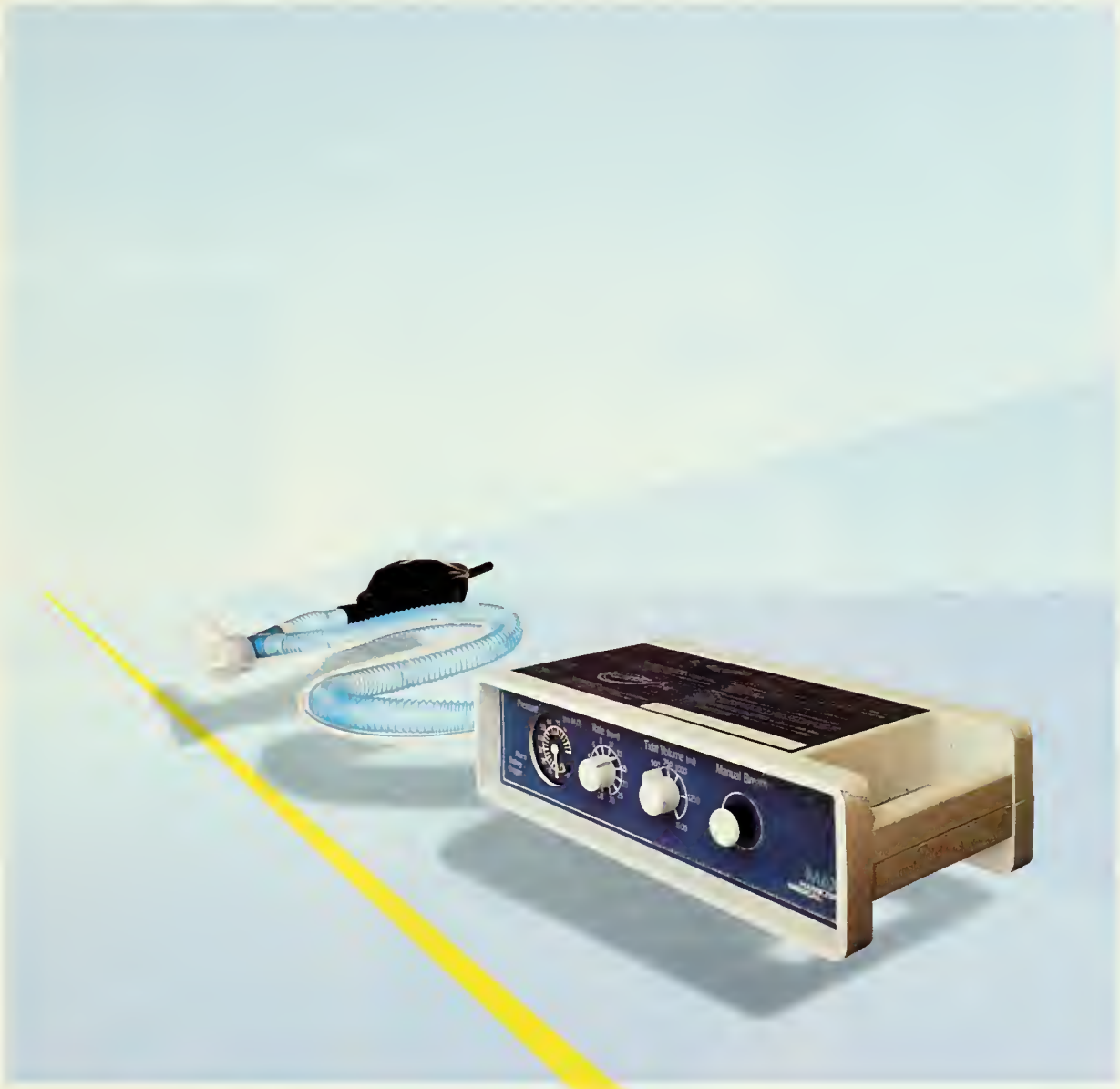

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Great Buys

Proclaim Your Feelings Of Pride And Professionalism To Your Community And Hospital

A Balloons

All children and adults love a balloon, and that makes these colorful balloons ideal for giveaways or for brightening up your celebration or health fair. Use them to proclaim *"Respiratory Care . . . Cares About the Air You Breathe"* or to add festivity to your booth or room decorations. Each package includes a variety of colors to make decorating fun.

Item R1 — $20, Pkg 100
(Member $10)

B Buttons

Wear them with pride and tell everyone you care with *"Respiratory Care . . . Cares"* buttons. Wear them during visits to schools for health promotion activities or on recruiting visits to respiratory care programs. After your presentation, distribute these colorful buttons, with a safety clasp, as mementos of your visit.

Item R6 — $14, Pkg 50
(Member $7)

C Respiratory Care Poster

Make a bold statement about respiratory care with this eye-catching color poster. Ideal for display in your department, cafeteria, or at health fairs. It proudly proclaims that respiratory care practitioners care about and for their patients.

Item R12 — $8 Ea
(Member $4)

D "Super RT" Brings Health Promotion to Life in Your Hands

Join forces with Super RT and the Smokebusters Club and bring health promotion to children. Wear your Super RT t-shirt and use puppets and skits to entertain and teach children about caring for their lungs. Each kit contains a Super RT t-shirt, three puppets, two scripts, 30 activity books, a lesson plan for presentations, and suggestions for future projects. Plus, each kit contains 30 FREE non-smoking fans, coloring books, and stickers.

Item PR7 — $40, Complete Kit (Member $30)
Item PR8 — $12, 10 Extra Activity Books (Member $8)

Great Buys

E Greeting Cards

This humorous set of four different greeting cards is great for your colleagues, patients, or friends with sayings such as, "I may be attached to my ventilator . . . but you are my inspiration!" Four-card set contains a birthday, get-well, friendship, and stop-smoking card.

Item R7 — $4, Pkg 4
(Member $2)

F Non-Smoking Fan

This hand-held fan is always popular at health fairs, and open houses. A great conversation piece or demonstration of one's commitment to better breathing. New-color fan helps you make the statement, "I'm a non-smoking fan."

Item R8 — $10, Pkg 25
(Member $5)

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Up to $15	$ 2.25
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$31-$50	$ 5.00
$51-$80	$ 6.50
$81-$100	$ 8.25
$100 and up	$11.00

G Health Tips Poster

This 15" x 22" poster will not only catch your eye, but it also gives pointers on respiratory health as well. Provides tips on dealing with air pollution, allergies, and asthma. Ideal for your department, health fair, mall display, school, or a doctor's office.

Item R42 — $8 Ea
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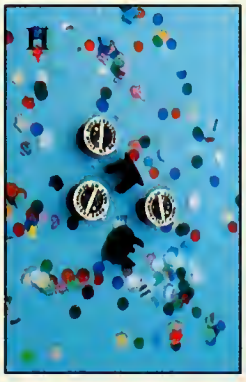

H No-Smoking Plugs

Advise passengers that there is "no smoking on board" with these auto cigarette lighter replacement plugs. Simply remove the lighter plug from your car and replace it with the no-smoking plug. Give them to your friends, colleagues, or patients to help them clear the air in their cars. *(Does not alter car. No tools required to install.)*

Item R11 — $12, Pkg 25 (Member $5.50)

I Headless Matches

Use them to help your smoking friends see the light and spread the word that "There's No Match for Respiratory Health" with these non-lighting headless matches to prove it. A perennial favorite in a bright new color.

Item R9 — $5, Box 50
(Member $2.50)

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Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

AARC SUMMER FORUM

The Registry, Naples, Florida, July 24-26, 1992

AARC ANNUAL CONVENTION SITES & DATES

1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994—Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5
1996—San Diego, California, November 2-5

OPEN FORUM 1992

It's time to submit your abstracts for possible presentation at the OPEN FORUM during the AARC Annual Meeting in San Antonio, Texas, December 12-15, 1992.

An early deadline (March 18) allows opportunity for revision following review, with re-submission by the final deadline (June 6). See Page 286 of this issue for more information

THE NATIONAL BOARD FOR RESPIRATORY CARE—1992 Examination and Fee Schedule

CRTT Examination

EXAMINATION DATE	MARCH 14, 1992
Applications Accepted Beginning	November 1, 1991
Application Deadline:	January 1, 1992
EXAMINATION DATE:	JULY 18, 1992
Applications Accepted Beginning:	March 1, 1992
Application Deadline:	May 1, 1992
EXAMINATION DATE:	NOVEMBER 14, 1992
Applications Accepted Beginning:	July 1, 1992
Application Deadline:	September 1, 1992

RRT Examination

EXAMINATION DATE:	JUNE 6, 1992
Applications Accepted Beginning:	December 1, 1991
Application Deadline:	February 1, 1992
EXAMINATION DATE:	DECEMBER 5, 1992
Applications Accepted Beginning:	June 1, 1992
Application Deadline:	August 1, 1992

RPFT Examination

EXAMINATION DATE:	DECEMBER 5, 1992
Applications Accepted Beginning:	July 1, 1992
Application Deadline:	September 1, 1992

CPFT Examination

EXAMINATION DATE:	JUNE 6, 1992
Applications Accepted Beginning:	December 1, 1991
Application Deadline:	April 1, 1992

Perinatal/Pediatric Examination

EXAMINATION DATE:	MARCH 14, 1992
Applications Accepted Beginning:	July 1, 1991
Application Deadline:	November 1, 1991
Application Fee:	$150
Reapplication Fee:	$130

Fee Schedule

Entry Level CRTT—new applicant:		$ 90.00
Entry Level CRTT—reapplicant:		$ 60.00
RRT Written and Clinical Simulation—new applicant:		$190.00
Written Registry Only—new applicant:		$ 90.00
Written Registry Only—reapplicant:		$ 60.00
Clinical Simulation Only new and reapplicant		$100.00
Entry Level CPFT new applicant:		$100.00
Entry Level CPFT—reapplicant:		$ 80.00
Advanced RPFT—new applicant:		$150.00
Advanced RPFT—reapplicant:		$130.00
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RRT Recredentialing.		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
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Reference: 1. Data on file, HealthScan Products Inc., Cedar Grove, NJ.
**U.S. Patent No. 5,040,527*

RC— 3/92

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1992 Call for Abstracts

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in San Antonio, Texas, December 12-15, 1992. Accepted abstracts will be published in the November 1992 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications—READ CAREFULLY!

An abstract may report (1) an **original study**, (2) the **evaluation of a method or device**, or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. A **method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience *or* a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. A **case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. First line of abstract should be the title. Title should explain content. Type or electronically print the abstract *double-spaced* on *plain white bond paper*, on *one page* only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a 1/2-inch left margin, and an approximate 1/2-inch ragged-right margin. Text submission on diskette is encouraged but must be accompanied by a hard copy. No identification of authors or institutions is to appear on the abstract sheet or within the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form *provided the table width is limited to 55 letter spaces* (ie, letters or numbers plus necessary blank spaces = 55). *No* figures or illustrations are to be attached to the abstract. Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 6 (postmark). Authors will be notified of acceptance or rejection by *letter only*— to be mailed by August 15. Authors may choose to submit abstracts early. Abstracts received by March 18 will be reviewed and the authors notified by April 24. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 6).

Mailing Instructions

Mail (Do not fax!) 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

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8. ❑ School

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C. ❑ Anesthesia Service
D. ❑ Emergency

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6. ❑ Home Care/Rehab
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